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PULMONARY VENTILATION AND ANESTHESIA IN MAJOR CHEST SURGERY

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SINCE major operative procedures inside the thoracic cavity were first tried, it is known that observations concerning altered respiratory conditions were made during these procedures and have been reported. The importance of these altered conditions has been emphasized and the danger of an acute wide unilateral chest opening has been pointed out. It was soon discovered that the respiratory difficulties were caused by alterations in the physiologic intrathoracic pressures. Due to the work of Sauerbruch in Germany and Rudolph Matas and Willy Meyer in this country, and others, methods were developed in order to correct these respiratory alterations. Their work resulted in the construction of negative pressure chambers, constant flowing positive pressure gas anesthesia machines, and rhythmically working respiratory pumps, of which only the constant-flowing positive pressure machines were constructed in such a way that they were suitable to apply for clinical use. Various types of these machines soon spread all over the world, and in connection with the use of them, thoracic surgery rapidly developed. However, the results of major intrathoracic procedures were not satisfactory, especially in cases in which prolonged operations were necessary and in which the pleura and mediastinum were normal at the time of operation. In order to get better results, these time-consuming operations were divided into stages. Because of multiple stages, the treatment was very considerably prolonged. Each stage had its percentage of postoperative complications, and the results were still not satisfactory.

Read before the Twenty-Second Annual Meeting of The American Association for Thoracic Surgery at Los Angeles, Calif., July 5, 6, and 7, 1939.

From Sabbatsberg Hospital, Surgical Clinic II, Stockholm. Surgeon-in-Chief, K. H. Giertz.

There are many factors other than ventilation and anesthesia that are of importance in improving our results in intrathoracic surgery. These include the difficulties in selection of the cases, difficulties in meticulous and exact technique to avoid intrathoracic postoperative hemorrhage which perhaps is more apt to occur in this region than anywhere because of a relatively high normal negative pressure in the pleural cavity and the mediastinum, difficulties in the infected cases in preventing spread of a local infection in a huge operative field, and the great difficulties in the safe closure of major bronchi when operating upon the lungs. These difficulties, I believe, are, from a technical point of view, the main problems which are not yet settled. However, to take up all of these problems for discussion would take too long a time, and I am going to discuss only our results and our views of the ventilation and anesthesia problems in major thoracic surgery cases.

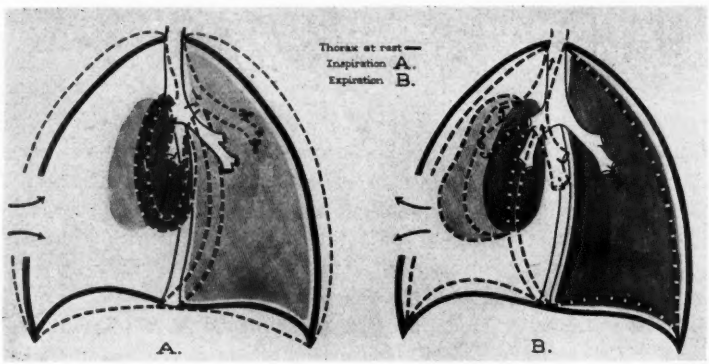

Fig. 1.

Fig. 1 *A* shows the well-known alterations in the physiology of respiration which take place with a wide-open chest on one side. The diagram shows a flow of gas, during the inspiratory phase of respiration, from the outside through the trachea and into the lung on the unopened side due to the expansion of thorax and the increased negative pressure within this lung. But, at the same time, as soon as the intrapulmonary pressure on the unopened side becomes more negative, one also finds a flow of the gas from the lung on the opened side into the lung on the unopened side. Because of this same change of the pressure inside the unopened lung, there is, during this period, a movement of the mediastinum to the unopened side, more or less decreasing the volume of gas flowing into that lung. This results in a low tidal volume and an impaired gas exchange. During the expiratory phase (Fig. 1 *B*) there is an increase of the pressure, that is, a less negative or positive pressure, in the lung on the unopened side, which is due mainly to the contraction of the expiratory muscles. This increased pressure blows the gas out of

the lung. When the gas passes the carina, a part of it flows back into the lung on the opened side where the pressure is still reduced and a part of it passes out through the trachea. The result of this, combined with paradoxical movement of the mediastinum, is that, at the same time that the patient is allowed to breathe only with one lung, his possibility of breathing with that lung is decreased. If one endeavors to correct these altered respiratory conditions by using an ordinary constant-flowing positive pressure machine, the mediastinum will be stabilized within certain limits by the positive pressure, but the flow of this so-called pendulum gas between the lungs continues. My impression is that the amount of the pendulum gas is certainly greater than is generally believed. In cases that I have observed, I have been astonished to see how large these gas volumes really are. I remember especially one case with a large chronic pulmonary abscess in which a lobectomy was done with the use of a constant-flowing positive pressure machine. The large closed abscess cavity collapsed completely during each inspiratory phase and was filled with gas during the expiratory phase. The volume of the cavity I estimated to be at least 75 c.c. The tidal volume in this case varied between 150 c.c. and 200 c.c. during the time the chest was wide open. Each inspiratory quantity of gas, in this case, was conclusively diluted by gas from the more or less collapsed lung on the opened side. This latter gas already had a low oxygen percentage and a high percentage of carbon dioxide, and its volume was about 50 per cent of the volume of the gas given by the anesthesia machine for each inspiration.

The ordinary positive pressure possesses another disadvantage in that it increases the difficulties of expiration. As you know, the expiratory muscles are not very well developed in man. If one tries to expire for a considerable length of time against slight positive pressure, he will soon find himself tired. I have found that expiratory difficulty gives an increase of carbon dioxide in the blood and acidosis after a certain length of time as a result of gradually increasing expiratory deficiency. The expiratory muscles try for as long a time as possible to overcome the increased expiratory pressures, but after a varying length of time in different persons, they are unable to fulfill the requirements for proper expiration, even when low positive pressure is used. When the expiratory muscles are becoming exhausted, the patients breathe with a more and more expanded chest cage. In order to diminish these expiratory difficulties, it is generally considered that as low a positive pressure as possible should be used, but, on the other hand, with a low positive pressure there is more extensive paradoxical motion of the mediastinum.

If the tidal volume is not sufficient, an increasing anoxemia, as well as an increase of carbon dioxide in the blood, develops. I am sure that with this method of breathing, as well as with spinal anesthesia and

oxygen given with or without positive pressure, the patient is bordering on a very small margin of safety because of poor ventilation.

The result of an impaired expiration has been studied very little. I have done a series of animal experiments in this respect and will now report two typical results in normal dogs. An airtight-fitting tube was inserted in the trachea. The inside diameter was 14 mm. so that there was no constriction of the airways. The tube was connected to an ordinary constant-flowing gas anesthesia machine. The chest was opened wide through an intercostal incision in the fifth intercostal space from the sternum to the spine. Positive pressure, varying between 4 and 8 cm. of water, was established as soon as the chest was opened. A pneumothorax was not allowed to develop on the other side. The curve in Fig. 2 shows that, during a period of a little more than three hours, in spite of giving the animal pure oxygen to breathe and in spite of a normal oxygenation of the blood, the carbon dioxide slowly but constantly rose in the arterial blood from the normal value of about 40 volumes per cent up to, in the first case, about 82 volumes per cent, and in the second case, to 95 per cent. The percentage of oxygen in the arterial blood was running at a normal level of about 20 volumes per cent during the experiments. After about three hours had elapsed in

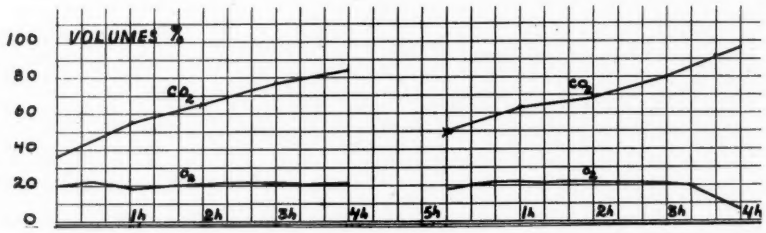

Fig. 2.

Experiment 2, the poisonous effect of the increase of carbon dioxide on the heart rapidly caused such a decreased circulation that an anoxemia developed in conjunction with irregular and weak heart movements. This is typical of carbon dioxide poisoning. If there is no anoxemia at the same time, there are almost no symptoms other than a change in the heart movements until a sudden heart collapse occurs. The change in the heart movements consists of a much more marked diastolic dilatation. There is only a slight change in blood pressure, but most often there is an increase in pulse rate.

These animals were under intravenous eunarkon anesthesia (an evipan preparation). In order to show that this anesthesia was not the cause of these alterations of the blood gases, the experiments were repeated with ordinary oxygen ether anesthesia. The curve obtained showed an oxygen and carbon dioxide curve of exactly the same type.

In 1912, while working with Sauerbruch in Munich, the chief of our department, Dr. K. H. Giertz, whom many members of the association will perhaps remember from his visit to their meeting in Rochester, observed the above-mentioned respiratory difficulties when using positive pressure in intrathoracic operations. In the years 1913-1916 he performed a number of experimental intrathoracic operations in an endeavor to find a method to correct these difficulties. The result of his work was published only in Swedish, and because of this, it is not well known. He came to the conclusion that rhythmic insufflation of gas was the safest method to use when operating with a wide-open chest cavity. To get the best results, he found that it is necessary to have an airtight intratracheal tube capable of transmitting physiologic quantities of gas for each breath and with a respiratory rate within normal limits. He also advised that the insufflated gas should be about body temperature and saturated with moisture. The maximum pressure during insufflation must not be allowed to go too high. Pressures up to 15 cm. of water were tried, and in more than one hundred experiments, there were no respiratory complications due to the method.

These investigations have been taken up by me during the years 1933-1939, both experimentally and clinically. I have been able to confirm all the results of Giertz. It is quite obvious that this type of insufflation is the method most similar to normal breathing when working with a wide-open chest cavity. A very great advantage of the method is that there are no alterations in ventilation, blood pressure, heart action, or general condition, should a bilateral wide-open pneumothorax develop, for example, during a difficult dissection in the mediastinum.

The ventilation anesthesia machine which Mr. Anderson, who is an engineer, and I have now completed and which I will describe in detail works in such a way that, regardless of a unilateral or bilateral opening of the chest, it is possible to give physiologic quantities of gas during the inspiratory phase, and it also allows the lungs to empty themselves without any resistance during the expiratory phase. The length of inspiratory and expiratory phases, that is, the respiratory rate and the maximum and the minimum pressures, can be controlled by adjustments in the machine. In order for the machine to take over the patient's breathing and to control the respirations, we have found that a certain amount of hyperventilation is preferable. As a result of moderate hyperventilation, the carbon dioxide content of the blood is decreased, and because of this fact, the irritability of the respiratory center is likewise decreased. When the respiration is under control of the machine, the movement of the respiratory muscles subsides. This is, in my opinion, another advantage of the method. In a majority of cases which I have had the opportunity to observe in different clinics in Germany,

England, and in this country, there has been a considerable increased work produced by the respiratory muscles during operations with a wide-open chest cavity. These respiratory movements of the normally working respiratory muscles, the intercostal muscles, the diaphragm and abdominal muscles, have been in many cases increased to such an extent that they must have been exhausting. In some cases I have observed that the auxiliary respiratory muscles have been working as well. If you try, as I have done, to use your respiratory muscles in a way such as can be observed in a great many of these operations, you will find that it is very uncomfortable and tiresome after only a short period of time and very difficult to go on with for even one-quarter of an hour. This type of strained respiration is, as far as I can understand, in most clinics considered to be more or less normal under these circumstances. In my opinion, however, the strained respiration adds considerably to the effect of the impaired ventilation that is produced by breathing against positive pressure, in that the strained respiration involves considerable muscle work which in turn consumes oxygen and produces carbon dioxide. If the patient is in the condition of *absolute muscular rest,* he needs a less amount of oxygen, and it is possible to carry on with a lower percentage of oxygen in the anesthetic gas mixture. In providing a high tidal volume at a moderately increased respiratory rate which can easily be done when using controlled respiration produced by a machine that gives rhythmic insufflation and free expiration, the oxygen percentage in the gas mixture can be further reduced without the slightest sign of anoxemia. In the majority of our cases in which we have used our machine, we have been able to use between 13 and 25 per cent of oxygen. In a few cases the amount of oxygen was brought down to about 10 per cent to produce sufficiently deep anesthesia with only a slight degree of cyanosis and much less cyanosis than in a great many cases I have observed operated upon with ordinary positive pressure and under ether, cyclopropane, or chloroform nitrous oxide anesthesia. This fact permits us to use nitrous oxide as a general anesthetic for the greatest part of the operation with sufficient, although slightly reduced, oxygenation. The general anesthesia that is produced is, however, not sufficiently deep to provide relaxation. By adding local anesthesia of the intercostal nerves, there is no reaction on opening and closing of the chest wall incision despite the superficial general anesthesia. Also, during the dissection of the mediastinum most of the reflexes can be sufficiently depressed by local anesthesia of the vagus, sympathetic, and phrenic nerves. During this part of the operation, however, the anesthesia produced by nitrous oxide most often is too slight. Because of this we have had to add small amounts of other anesthetics, among which we prefer cyclopropane. In only a very few cases in which we have been able to reduce the oxygen sufficiently, but without clinical signs of anoxemia have we been able to perform the

whole operation (such as total pneumonectomy) with only nitrous oxide oxygen and local anesthesia. If cyanosis is necessary for sufficiently deep anesthesia, we consider this an indication for adding more oxygen and an additional anesthetic.

We are of the opinion that nitrous oxide is the ideal anesthetic for intrathoracic operations, providing there is sufficient oxygenation. Nitrous oxide does not irritate the mucous membranes of the respiratory tract. It is very rapidly absorbed and expelled, and it has no lasting poisonous effect. It has no depressive influence on the cardiovascular system, and it is the least explosive general anesthetic gas mixture that is now used. This allows the opportunity of using the electrosurgical cautery during the most part of the operation. It is only during the short periods that we add cyclopropane and a few minutes afterward that we do not dare to use the diathermy cautery, which we prefer. We consider the diathermy cautery a very valuable means to control bleeding from the chest wall when dividing adhesions and to diminish the risk of a spread of infection when dividing bronchi or the esophagus.

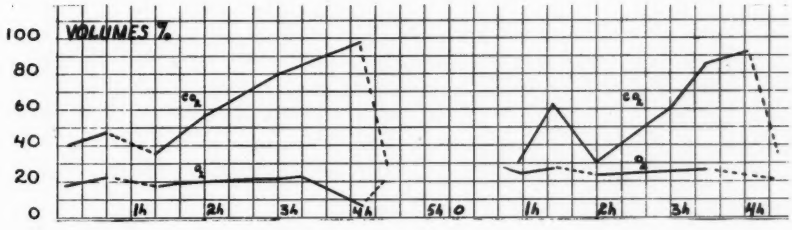

Fig. 3.

This combination of anesthesia and ventilation gives, as I have already said, no resistance to expiration. This is very important and secures a normal or, in case of slight hyperventilation, a moderately decreased carbon dioxide content in the blood. Fig. 3 shows how the high carbon dioxide content, which is produced by breathing against slight positive pressures produced by an ordinary constant-flowing positive pressure anesthesia machine during the time the chest is wide open, is rapidly decreased to slightly subnormal values by changing to the rhythmic ventilation and is again increased when turning to ordinary slight constant-flowing positive pressure breathing.

I have observed that rhythmic insufflation seems to increase the anesthetic effect of nitrous oxide without decreasing the oxygen to such an extent that clinical signs of anoxemia occur. I believe that this observation is due to the fact that, with our machine, we are using considerably higher tidal volumes than are required by the patient. We force the lungs to function in a manner that they normally would when a larger amount of oxygen is needed by the body, such as in a nonresting state or in mild exercise. This type of breathing, i.e., an in-

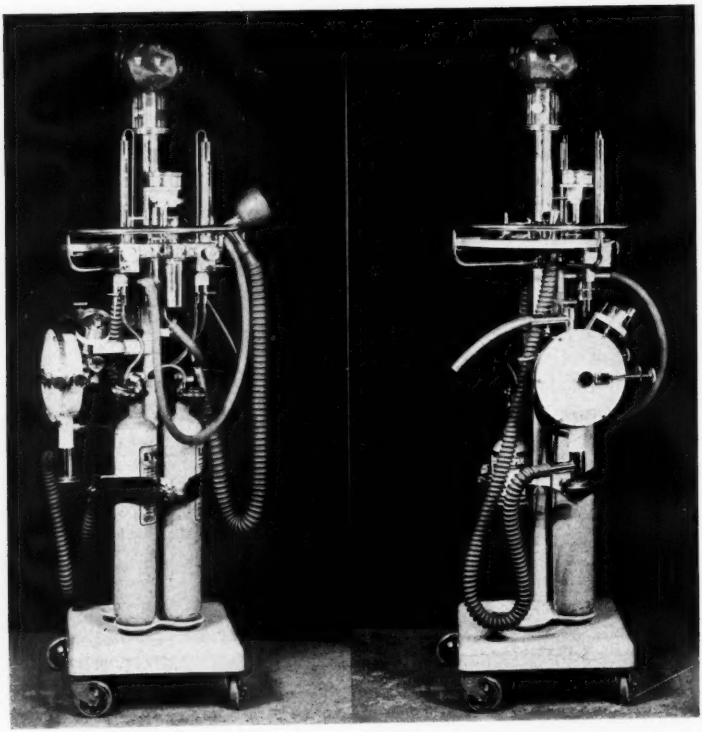

Fig. 4.

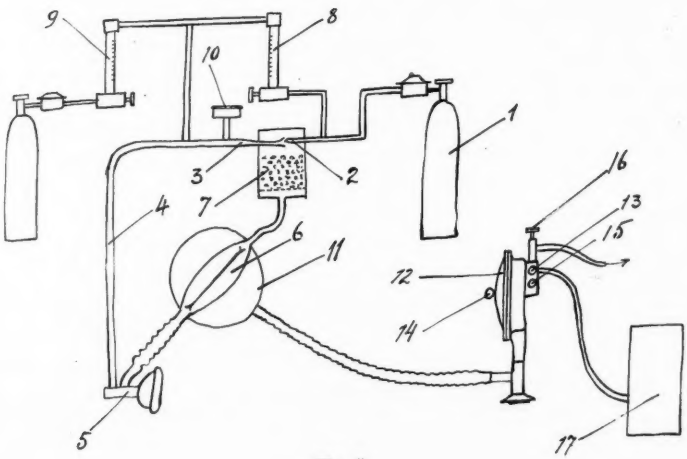

Fig. 5.

creased tidal volume and a slightly increased rate, allows us to provide a sufficient volume of oxygen per minute to the resting body in spite of a high nitrous oxide percentage and a decreased oxygen percentage in the gas mixture. This observation is in accordance with the theory of breathing that is now favored more and more by many physiologists in different countries, such as Best in Canada, Kramer and Sarre in Denmark, Sjostrand in Sweden, and Waters in the United States. This theory is that when the body is at rest, only certain areas of the lung are in use because only small amounts of oxygen are required and only small amounts of carbon dioxide are produced. It seems logical to me to assume that, if the lungs can be forced to function as if a relatively high amount of oxygen were required when it is not, it is possible to cover the oxygen need with a lower percentage of oxygen in the inspired gas mixture at the same time as the required high percentage of nitrous oxide, for example, can be administered to give superficial anesthesia.

The apparatus we now use consists of a combination of a modified Frenckner spiropulsator and a continuously flowing gas narcosis apparatus built according to a new principle (Fig. 4). The different parts of the new anesthesia machine are indicated by the numbers *1* to *10* and the simplified Frenckner machine by the numbers *11* to *17* in Fig. 5.

From the container *1* the nitrous oxide flows through the jet *2* into the Venturi tube *3*. Because of the kinetic energy of the gas flow, ten times as much gas is sucked through the soda lime container *7* into the Venturi tube *3*. This gas mixture flows further on through the tube *4* to the face piece *5*, and if this is closed, back to the rubber bag *6* and to the soda lime container *7*. Thus, we have obtained a gas flow automatically circulated through the soda lime. The rapidity of the circulating gas is about ten liters per minute when the amount of gas passing through the jet *2* is regulated to a flow of one liter per minute. A gas flow like this reduces the dead space practically to zero; that is, we provide the face piece with fresh gas from the very beginning of and throughout the whole inhalation phase. If an increased flow of nitrous oxide is required, as for example at the beginning of the anesthesia, this is provided for through the flow meter *8*. The oxygen flow is regulated by the flow meter *9*. In case ether-oxygen or cyclopropane-oxygen anesthesia is preferred, oxygen is forced through the jet *2* and Venturi tube *3* instead of nitrous oxide, and cyclopropane or ether is added at *10*. Naturally cyclopropane or ether can be added if required during a nitrous oxide anesthesia.

When operating with a wide-open chest, the rubber bag *6* is enclosed in a glass container *11*. The glass container is connected to a simplified spiropulsator *12* of the same principle that Frenckner described. The spiropulsator rhythmically increases and decreases pressures inside the glass container *11*, thus compressing and releasing the rubber bag *6*,

providing rhythmic artificial respiration. The spiropulsator receives compressed air from the electrically driven compressor *17*.

Through simple regulation of the different hand wheels, *13*, *14*, *15*, and *16*, respectively, the length of the inspiratory phase, the length of the expiratory phase, the maximum pressure, and the minimum pressures are controlled.

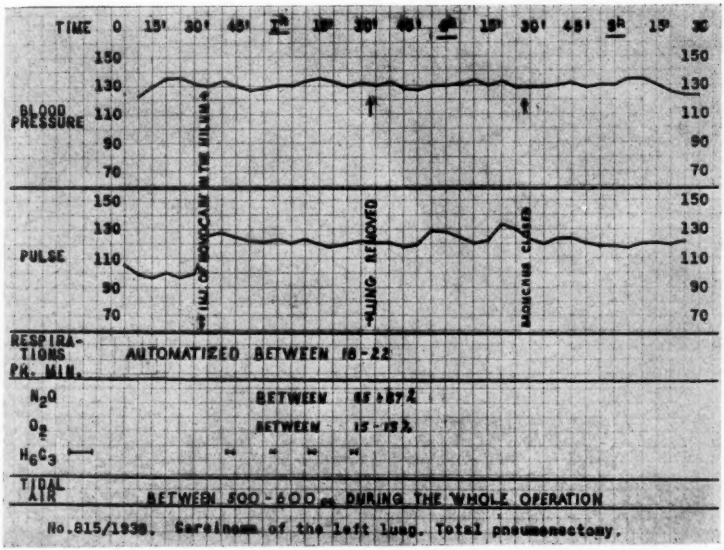

Fig. 6.

I will now give a brief report of the actual management of a typical case, a case of a squamous cell carcinoma of the left lower lobe bronchus growing up to the level of the upper lobe bronchus in a man 50 years of age. He was given the usual premedication of atropine, codeine, and morphia that we give to a male adult in good condition and weighing about 170 pounds, that is, three-quarters of an hour before the anesthesia, ⅓ gr. morphia and ⅓ gr. codeine of phosphate. One-half hour before operation we give 1/60 gr. of atropine sulfate and another ⅓ gr. of codeine phosphate. One-quarter of an hour before operation we give ⅙ gr. of morphia and another ⅓ gr. of codeine phosphate. The patient is usually quiet and sleepy, but not dopey. Then the pharynx, larynx, trachea, and bronchi of the diseased side are anesthetized with 2 per cent pantocaine mixed with adrenalin, as before a usual bronchoscopy. In infected cases the patient is then bronchoscoped, all secretions are aspirated, and the bronchus of the diseased side is blocked with a gauze tampon moistened with 2 per cent pantocaine. The strip of gauze used is 1 cm. wide and between 50 and 125 cm. in length, depending upon the width and the length of the bronchus we want to block.

For a lobectomy only the bronchus to that lobe is blocked; and for a total pneumonectomy, the entire bronchus up to about 1 cm. from the carina. This gives the advantage of a prolonged local anesthesia of the interior of the bronchus which is to be dissected. The tamponage gives security so that no secretion is squeezed out of the diseased lung into the bronchial tree during operation, and we consider this to be of importance. A thin silk thread is attached to the end of the tampon. The silk thread is then brought out through the bronchoscope and is passed through a metal spiral which in turn is attached to a thin steel probe. The spiral and the probe are introduced into the bronchoscope along the silk thread and down to the top of the tamponage. The bronchoscope is removed; the steel probe with its attached spiral remains in place and is gently pressed against the top of the tamponage so that the tamponage cannot be displaced by the respiratory movements or the manipulation of the bronchus during the operation. Before using this steel probe we once had trouble because a part of the tampon moved over in the other bronchus, causing partial obstruction, and had to be removed. An intratracheal tube* with an inflatable balloon is then passed into the

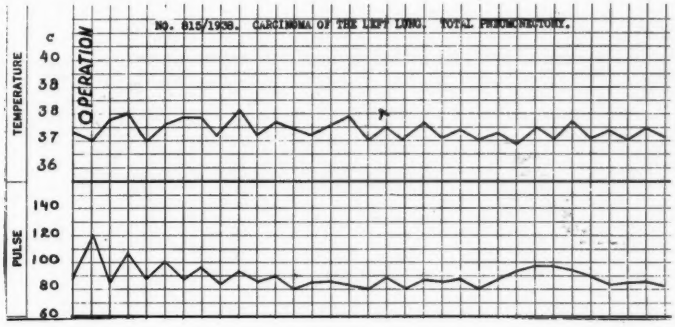

Fig. 7.

trachea in such a manner that the steel probe remains inside the tube. The length of the steel probe can be adjusted by a screw mechanism and is shortened or lengthened so that the end of it which comes out of the intratracheal tube stands against the inside of the metal piece connecting the anesthesia machine and the intratracheal tube. The inside diameter of this tube must be at least 10 to 12 mm. for an adult. The general anesthesia is then started. In the beginning the patient is allowed to breathe of his own accord a mixture of nitrous-oxide-oxygen in which the oxygen is carried so low that a very slight cyanosis is permitted to take place. After about ten minutes the rhythmic ventilation is started. Small quantities of cyclopropane plus oxygen are usually added, as the anesthesia is most often not sufficiently deep to allow controlled respira-

*It is certainly important to have a tube that does not give much more resistance to the gas that passes than does a normal airway. We have been working on the construction of an ideal tube, but this problem has not yet been solved.

tion; that is, the patient's respirations are automatized and controlled by the machine. Before this stage is reached, it is necessary to regulate the inspiratory and expiratory phases manually, following the patient's own inspiratory and expiratory movements. After a few minutes the machine takes over the breathing of the patient and the rate of respiration, the tidal volume, and the pressures can then be adjusted as required. (Motion picture film demonstrated.) The anesthesia chart

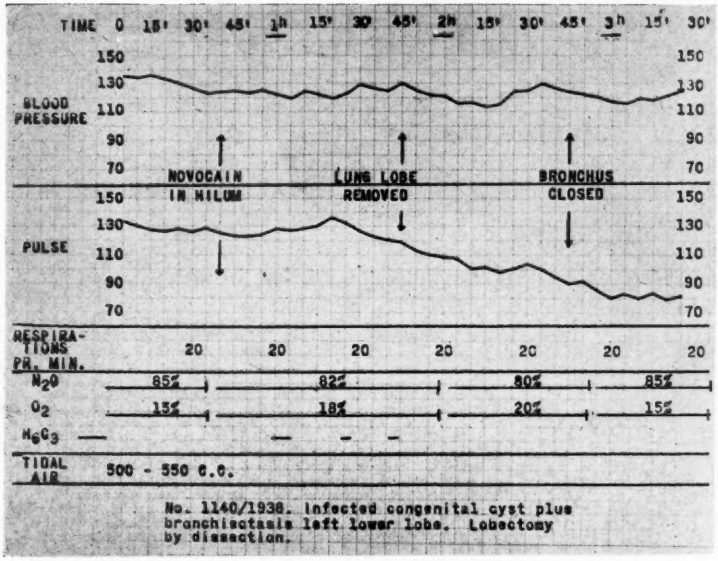
TIME 0 15' 30' 45' 1h 15' 30' 45' 2h 15' 30' 45' 3h 15' 30'
BLOOD PRESSURE
150
130
110
90
70
NOVOCAIN IN HILUM
LUNG LOBE REMOVED
BRONCHUS CLOSED
PULSE
RESPIRATIONS PR. MIN.
20 20 20 20 20 20 20
N_2O 85% 82% 80% 85%
O_2 15% 18% 20% 15%
H_5C_3
TIDAL AIR 500 - 550 c.c.
No. 1140/1938. Infected congenital cyst plus bronchiectasis left lower lobe. Lobectomy by dissection.

Fig. 8.

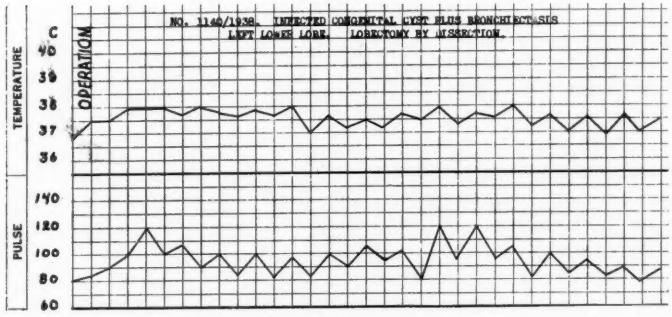

Fig. 9.

(Fig. 6) of this particular patient shows the blood pressure, the pulse frequency, the respiratory rate, and the time when cyclopropane was required. In Fig. 7 is shown the temperature chart of the patient postoperatively. Figs. 8 and 9 show the same findings in one case of infected

bronchiectasis and congenital cyst of the left lower lobe, operated upon with lobectomy by dissection technique. Figs. 10 and 11 are charts of a case of mediastinal tumor removed transpleurally.

We have now had ninety-four intrathoracic operations in which we have used rhythmic ventilation in connection with inhalation anesthesia.

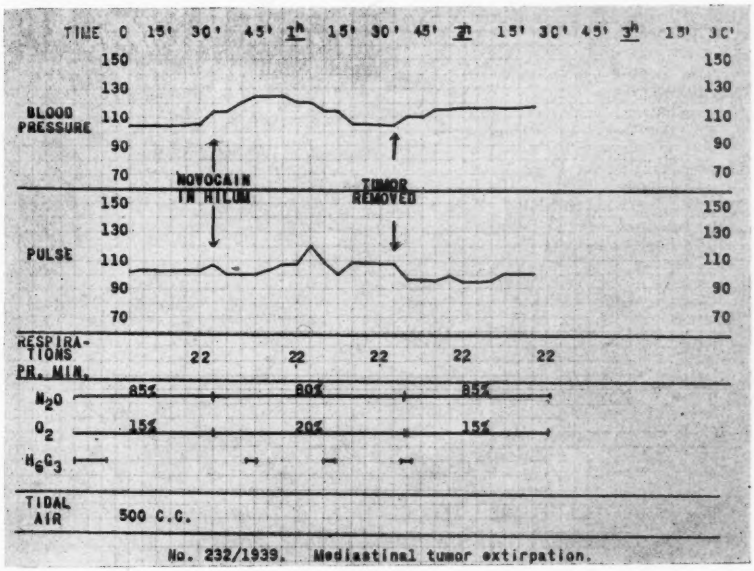

Fig. 10.

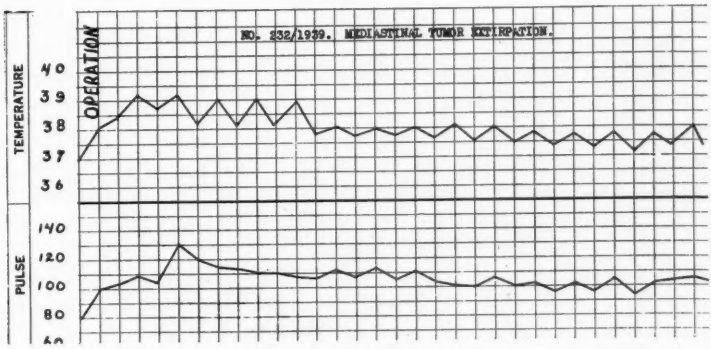

Fig. 11.

These cases belong in the groups as seen in Table I. In this group of ninety-four cases, there have been twenty-seven deaths, two of which occurred during operation. In one of the patients an inoperable lung carcinoma, as it turned out, was explored for, which at autopsy was

TABLE I

NUMBER OF CASES OF INTRATHORACIC OPERATIONS WITH AUTOMATIZED BREATHING (94)

Bronchial carcinomas	
Total pneumonectomies	19 (10 postoperative deaths)
Lobectomies	2 (1 postoperative death)
Exploratory thoracotomies	25 (6 postoperative deaths)
Benign bronchial tumor (bronchioma)	
Lobectomy	1 (Recovered)
Bronchiectasis	
Total pneumonectomies	1 (1 postoperative death)
Lobectomies	17 (4 postoperative deaths)
Tuberculous bronchial strictures with pulmonary tuberculosis and secondary bronchiectasis	
Total pneumonectomies	4 (Recovered)
Mediastinal tumors	
Extirpation	6 (Recovered)
Peripheral intrabronchial foreign bodies	
Transpleural bronchotomies	4 (Recovered)
Lung echinococcus	
Pulmonary resection	2 (Recovered)
Carcinoma of the esophagus	
Total esophagectomies	4 (4 postoperative deaths)
Tuberculous osteochondritis	
Block chest wall resection	2 (Recovered)
Intrathoracic thyroid adenomas	
Extirpation	5 (Recovered)
Operations on pericardium and heart	
Pericardial resection	1 (1 postoperative death)
Heart stab wound	1 (Recovered)
Total	94

found to be very soft and fragile. A small pulmonary vein inside the tumor had ruptured, and the patient died suddenly of air embolism. The left heart and all cerebral arteries were full of air. The second case had very distressing cough reflexes, difficulty with breathing, and slight cyanosis during the intratracheal and intrabronchial pantocaine anesthesia before the bronchial tamponage was inserted. When the bronchoscope was passed, there was a quantity of very thick and tenacious sputum in the right main bronchus, coming from the diseased middle lobe bronchus. In this patient the cause of the constriction of the middle lobe bronchus and the complete atelectasis of the middle lobe was not certain. He was operated upon because the clinical symptoms were those of a carcinoma of the middle lobe bronchus, although biopsy was never positive for carcinoma. He had been observed for two months in another hospital where they had found no evidence of tuberculosis. When the bronchial tamponage was inserted, he complained of shortness of breath, had very marked cough reflexes and paroxysmal attacks of cough, and his cyanosis did not disappear. At the time the tamponage was introduced, the left main bronchi were inspected and found to be empty, and the mucous membrane was normal. As we had never before had an experience like this one, we were doubtful whether we should continue with the anesthesia or whether we should postpone the operation. However, we decided that the probable explanation of

the symptoms was a very high irritability of the bronchi with marked reflexes which were not suppressed by the local anesthesia, and we hoped that when the general anesthesia was deep enough, the reflexes would vanish and the symptoms disappear. In this case we had to give between 40 and 50 per cent oxygen plus cyclopropane in order to get anesthesia and to diminish the cyanosis. When the chest was opened, he gradually became worse and the cyanosis increased, so we decided not to continue the operation but to complete it in another stage. However, his condition suddenly became worse, his heart movements became irregular, and after a few minutes the heart suddenly stopped. With intracardiac adrenalin and heart massage, the circulation was reestablished, and closure of the chest wound was started. When the muscles were being sutured, the heart again suddenly stopped, and it was then impossible to revive the patient. At the same time as the circulation ceased the first time, the bronchial tamponage was removed, and intrabronchial suction established without much effect. It seemed as if the major bronchi were empty. At autopsy the small bronchi beyond bronchoscopic visibility, both on the diseased and the good sides, were found to be obstructed by very thick material of the same kind as was aspirated from the main bronchus on the right side before the tamponage was inserted. So much of the pulmonary area was occluded that the cause of death was considered to be anoxemia because of too little functional lung tissue. The cause of the bronchial obstruction on the right side was a tuberculous lesion of a lymph node outside the middle lobe bronchus. This lymph node was destroyed and replaced by very thick caseous material. It had broken through the bronchial wall, and, in my opinion, spread of this content before intrabronchial tamponage was done was the cause of death.

Before I finish I will, in a few words, take into consideration the tracheobronchial pulmonary complications that could be caused by the anesthesia method used. We have had no demonstrable mechanical damages of any importance, such as ruptures of lung tissue or injuries of the mucous membranes. We have had five cases with extensive infection in the bronchial tree or lung tissue or both, and this infection must be considered as the main cause of death. In one case of total pneumonectomy for carcinoma, the autopsy showed a marked narrowing of the remaining main bronchus as a result of faulty technique in invagination of the resected main bronchus. In another case of total pneumonectomy for carcinoma, a previously undiagnosed infected bronchiectasis in the remaining lung was found at autopsy. In these cases I do not believe it is justified to consider the intratracheal and intrabronchial manipulation or the type of anesthesia as the cause of these complications. In the three remaining cases, one total pneumonectomy and two total esophagectomies, there was no reason at autopsy why these bronchopulmonary complications should have occurred, and it may be that

our type of anesthesia in these cases played some role in producing the complications. We do not think, however, that it can be considered as an abnormally high mortality rate to have about 3 per cent mortality due to bronchopulmonary infection in a group of cases such as this.

The general impression that we have of this type of ventilation and anesthesia is that it is much superior to any other type we have tried or seen used. It allows us an opportunity to work inside the open thoracic cavity without any hurry and without any disturbance from the point of view of oxygenation or disturbed carbon dioxide exchange, for any length of time we wish, and regardless of a unilateral or bilateral open pleura. This gives us time to do a meticulous dissection in the mediastinum and to spend sufficient time in cases of total or partial pneumonectomy to close the bronchi in the way I have described earlier. Although this method of closure is rather time-consuming, we have found it to be more reliable than any other type of closure we have seen performed or described. Through this type of regulation of the pressures while working inside the thoracic cavity, we have, from a technical point of view, the advantage that the mediastinum never moves over to the unopened side to the same extent as with ordinary positive pressures or with spinal anesthesia. Accordingly, the approach to the mediastinum is made much easier. In operations lasting from four to five hours this type of ventilation and anesthesia seems to prevent, to a certain extent, postoperative shock, and our patients are very seldom more affected now after a long intrathoracic operation than after an abdominal one of the same or even shorter duration. The only thing we have to follow closely is the circulation. Any failure can be easily corrected by giving blood or stimulants through the needle we always insert at the beginning of the operation for giving intravenous fluid continuously during the operation.

SUMMARY

A description of a new continuously flowing gas anesthesia machine provided with means for mechanically controlled respiration is presented.

The importance of mechanically controlled automatized breathing with rhythmic insufflation during the inspiratory phase and free outflow of gas during the expiratory phase during open chest operations is pointed out.

The automatized type of ventilation is the only method that gives a type of respiration which is similar to the normal breathing when the chest is wide open on one or both sides.

This type of ventilation makes it possible to decrease the oxygen in the anesthetic gas mixture without anoxemia to such an extent that nitrous oxide can be used as a basic general anesthetic agent. The

anesthesia produced is, however, too light without local nerve block of the chest wall and mediastinum. If necessary, cyclopropane can be added during short periods when deeper anesthesia is required.

Controlled automatized respiration under general anesthesia gives absolute rest of all the respiratory muscles and a quieter operative field than any other method.

A brief report of the results of this type of ventilation and anesthesia in the author's cases is given.

DISCUSSION

DR. J. STUART HARRINGTON, Rochester, Minn.—I congratulate Dr. Crafoord on his splendid presentation. I think that this is a definite advance in our anesthesia progress. I had the pleasure of seeing Dr. Crafoord in Stockholm four years ago when he was developing this machine for the administration of anesthesia. He used it very satisfactorily in two cases which I saw at that time. There are many factors which must be taken into consideration in anesthesia for chest surgery. I think it is of great advantage in intrapulmonary lesions not to traumatize the trachea, and, in many cases in which I perform a transpleural operation, I do not utilize intratracheal anesthesia but use a closed mask method, always having the intratracheal tube accessible to insert if it becomes necessary. I believe it is very advantageous to all types of surgery that more interest is being taken in the anesthetic agent and its administration. I am sure that thoracic surgery has stimulated more interest in this field than any other type of surgery. I believe thoracic surgery has been an important factor in bringing the general surgeon's attention to the important part that anesthesia plays in surgical procedures and recovery.

DR. CAMERON HAIGHT, Ann Arbor, Mich.—Dr. Crafoord is to be congratulated on his excellent paper. We are honored to have him here today to present his type of anesthesia which is a great advance for thoracic surgery. I have had occasion to see the machine used on two occasions, one in the case of a foreign body in the lung, which Dr. Crafoord removed in his own hospital. It was very interesting at the time when Dr. Crafoord was actually working on the lung to see the patient's respirations slowed to approximately five or 6 per minute. The other case was a lobectomy recently done in Ann Arbor, for which Dr. Crafoord kindly used his machine for the anesthesia. The lobe was rather densely adherent to the diaphragm. One of the points about the anesthesia that particularly impressed me was that the diaphragm remained absolutely quiet during the operation, and a difficult dissection of the lower lobe was greatly facilitated.

DR. EMILE HOLMAN, San Francisco, Calif.—I should like to congratulate Dr. Crafoord. I had the opportunity two years ago to see the development of this machine. At that time it was a magnificent affair. It covered at least 10 square feet of the operating room and required two doctors, two anesthetic engineers, and two nurses to run it. At the present time he has simplified it into an excellent machine. It can be run, as demonstrated in our clinic, by one man. I wish also to testify to the extraordinary facility with which one can operate under this anesthesia. One is not troubled in the least by respiratory movements or by the necessity of inflating the lung at frequent intervals.

DIFFERENTIAL PRESSURES AND REDUCED LUNG FUNCTION IN INTRATHORACIC OPERATIONS

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THORACIC surgery today enjoys a position similar to that occupied by the surgical specialties dealing with other parts of the body. Its rapid advancement has been chiefly during the past two decades. Its accomplishments have been many, but problems still remain regarding new points of attack as well as complications associated with operations now being performed.

Surgery of the chest has advanced to this position due to an increased knowledge of cardiorespiratory physiology, along with improvements in diagnostic methods. During the course of its evolution there have been many obstacles. Erroneous impressions gained through the lack of a thorough understanding of the function of organs within the chest have been of paramount importance in this respect. Examples of this may be seen in a brief review of its development.

The dangers of an open pneumothorax were well realized by the ancient Greeks. In the sixteenth century the anatomist Vesalius taught his students the hazards associated with this condition, but also demonstrated how they could be obviated or overcome once it was produced. The former was long remembered, but the latter was forgotten or, at least, not applied to clinical problems dealing with these structures until within the past three decades. In the early part of the twentieth century much attention was given to the subject of operations on the thorax, and the negative pressure chamber and positive pressure anesthesia were the fruits of this work.

With the further development of this branch of surgery, new fields were explored, and courage to perform unheard-of operations was gained through a sense of security obtained by the use of positive pressure anesthesia.

Progress in thoracic surgery has been so rapid during the past two decades that it has not permitted the development of a thorough understanding of the cardiorespiratory physiology to keep pace with the surgical accomplishments. During the World War numerous examples of the possibility of operating in the pleural cavity to remove missiles of various kinds gave rise to the erroneous impression that the danger of an open pneumothorax had been greatly overestimated. However, the tragic results accompanying early open drainage of acute streptococcal empyema complicating cases of influenza in 1917-1918

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were shown by Graham and Bell[1] in their physiologic studies to be the direct results of creating an open pneumothorax in the presence of a markedly decreased vital capacity.

It has long been known that one can open the thoracic cavity in some patients without ill effects and that some persons can survive an open wound in the chest wall. This marked discrepancy can be explained only in the light of altered physiology by pathologic changes within the chest.

The normal function of these organs may be altered by a variety of intra- or extrapulmonary lesions. Inflammatory conditions of the lungs and pleura may reduce the vital capacity 50 per cent or more, and yet, on physical or x-ray examination, the lung fields may appear to be relatively normal. Alterations of the tracheobronchial tree by neoplasm or inflammatory changes may likewise markedly decrease the amount of functioning lung tissue. Extrapulmonary conditions include not only the chest wall, mediastinum, and diaphragm, but also cardiac activity and the condition of the circulating blood as well. It may be readily understood that diminished cardiac output or marked anemia is much less well tolerated in the presence of a lowered vital capacity than when the lung function is normal.

These factors have a direct bearing upon intrathoracic surgery or in operations which decrease the amount of functioning lung. Thus, the importance of knowing:

First, (a) the vital capacity preceding operation, (b) the condition of the cardiac function, and (c) the condition of the oxygen-carrying function of the blood; second, the amount of functioning lung during operation and how it is best maintained; third, the amount of functioning lung remaining following operation.

The importance of the various factors mentioned above will vary, depending upon:

1. The expected diminution of lung function due to removal of or collapse of lung tissue,
2. The amount of blood loss during operation,
3. The amount of diminished respiratory effort, and
4. The effect of the operation upon cardiac output.

The vital capacity necessary to sustain life in healthy animals has been found to be definitely less than 50 per cent and is probably less than 25 per cent of the normal in most cases (Heuer and Andrus[2]). Investigative work in our laboratory has shown that not only can a healthy dog survive with but one lung lobe (an upper), but also that when quiet, it appears to be perfectly normal to external examination. The dog can tolerate this marked reduction in vital capacity only when it is effected over a relatively long period of time (three to six months). The right upper lobe represents about 15 per cent of the entire lung capacity.

Blood studies made during the reduction in vital capacity reveal an early rise in hemoglobin, hematocrit reading, and red cell count. These changes, however, gradually revert to the normal after a period of several months. The remaining lung lobe becomes greatly hyperinflated, the alveolar walls become torn, and a spontaneous pneumothorax may occur one or more times during the reduction in functioning lung. If the reduction in vital capacity is produced too rapidly, the animal dies of spontaneous pneumothorax and cardiac failure. A complete report of this work may be found elsewhere (Adams,[3] Phillips, Adams, and Hrdina[4]). In view of these findings, when clinical conditions require the removal of much lung tissue involving both sides of the chest, it should be much safer to do so in two or more operations separated by at least from three to six months. By knowing the vital capacity preceding the operation, some indication of the margin of safety is obtained, and thus an indication of how much functioning lung tissue can be resected as well as how much collapse of lung tissue during the operation can be allowed. The resection of only a part of two or more involved lobes in bronchiectasis, thus preserving all available functioning lung tissue as advocated by Churchill,[5] is a big step forward in broadening the application of the surgical treatment of that disease.

As in operations elsewhere in the body, exposure of the operative field may be of paramount importance. This is especially true in operations upon the esophagus and other mediastinal structures or upon the diaphragm. This is greatly facilitated by allowing the lung to collapse to a considerable degree. Ohsawa[6] at the Imperial University in Tokyo believes that the lack of adequate exposure due to the use of too much positive pressure in the administration of the anesthesia has been the greatest single detriment in the development of surgery of the esophagus. Untoward symptoms, such as dyspnea and cyanosis, during intrathoracic operations need not be due to insufficient inflation of the lungs. They may be due to decreased cardiac output from pressure on the great veins or on the auricles, or to overdistention of the lungs with increased resistance to the flow of blood through the lungs,[7, 8] or to decreased oxygen-carrying capacity of the blood. The importance of knowing the percentage of lung function and other factors influencing blood gas exchange preceding the operation is thus quite obvious.

ANESTHESIA

The maintenance of sufficient lung function during a surgical pneumothorax is of paramount importance to the success of the operation. Positive pressure anesthesia administered through an intratracheal catheter has enjoyed much success in performing this function. The lungs are maintained in an inflated condition, and "controlled respiration" may be obtained by the use of proper mixtures of anesthetic

agents under sufficient pressure. While performing the above function, it also prevents suffocation by spasm of the glottus and, furthermore, allows aspiration of mucus and pus from the tracheobronchial tree during or following the operation. Positive pressure intratracheal anesthesia has been so thoroughly accepted that insufficient attention has been given the accompanying hazards or disadvantages. These are ever present and occasionally lead to disastrous results, even in the hands of anesthetists properly instructed in the use of this apparatus and method (Stephens,[9] Bradshaw,[10] Bourne,[11] Heidrick et al.[12]). Elsberg[13] is usually credited with having performed the first thoracic operation using an anesthesia giving intratracheal insufflation, although it is known that Tuffier of Paris, in 1896, passed a metal tube through the larynx and anesthetized the patient in this manner, following which a partial lung resection was performed. In 1911 Elsberg reported more than 100 cases in which this type of administration had been employed with very satisfactory results.

Little investigative work has been reported regarding the effect of positive pressure upon the cardiorespiratory physiology in general and the parenchyma of the lung in particular. Eisenbrey,[14] in 1912, used intratracheal anesthesia in experimental work and concluded that the pressure in the tracheobronchial tree was little affected by the pressure with which gas or air was forced into the lungs.

Positive pressure anesthesia may be instituted through an intratracheal catheter or by the closed face-mask method. The types of apparatus for administering the former are many and varied, but in principle they are similar.

In investigative work on cats, Macklin[15] demonstrated the presence of mediastinal emphysema and pneumothorax following the use of intratracheal anesthesia. He reasoned that the mediastinal emphysema resulted from the tears of the alveolar walls, the air travelling along the vessels in the interstitial tissue to the root of the lung into the mediastinum, and that the pneumothorax was secondary to the emphysema. He was unable to demonstrate lacerated alveolar walls by stained microscopic sections, but did show interstitial emphysema in the perivascular sheaths. Wall pressures used varied from 10 to 220 mm. of mercury. Our interest in this question was stimulated by the death of several animals within twelve hours following intrathoracic operations. In these operations a low positive pressure anesthesia with an intratracheal catheter was employed, and at death complete collapse of the lungs was found. In our recent studies both cats and dogs were used, and the intrabronchial pressure was measured during the administration of the anesthesia. Pressures used varied from 1.0 to 110 mm. of mercury. The object was to find out what pressure was necessary to produce mediastinal emphysema and pneumothorax. Changes in

respiration and cardiac action were noted. A brief résumé of the results follows, a complete report of which may be found elsewhere.[16]

There was a routine fall in the arterial blood pressure of from 8 to 10 mm. of mercury, with a 6 mm. of mercury rise in intrabronchial pressure, and as much as 98 mm. of mercury fall in blood pressure, with 110 mm. of mercury rise in intrabronchial pressure. Intrabronchial pressures of 24 mm. of mercury or above were routinely accompanied by the development of a mediastinal emphysema in dogs. The pressure necessary to produce the same effects in cats was 20 mm. of mercury. Intrabronchial pressures which produced a fall in arterial blood pressure also caused a cessation of respiratory movements. This change persisted for from thirty seconds to several minutes or longer, depending upon the anesthetic mixture and intrabronchial pressure employed, thus creating a state of "controlled respiration."

That changes such as these occur clinically is seen in reports on this subject. Bradshaw[10] reported a case of a child in whom emphysema of the mediastinum, as well as about the neck and face accompanied by pneumothorax, resulted shortly after the beginning of an intratracheal anesthesia. The anesthesia was discontinued and a fatality prevented. From a personal communication, Wesley Bourne[11] knew of several cases of emphysema following positive pressure anesthesia. A similar case is being reported by Heidrick et al. of this clinic.[12] Stephens[9] reported postoperative pneumothoraces on the contralateral side in three cases following intrathoracic operations, which may quite easily have been due to this cause. A patient with mediastinal emphysema and subcutaneous emphysema of the neck and face following an asthmatic attack with severe coughing was observed in our clinic. The principle involved in production of the emphysema was undoubtedly the same as in the above cases.

These studies and this report are not intended to discourage the use of positive pressure anesthesia. On the contrary, a very low-grade positive pressure anesthesia is an important, as well as necessary, factor in performing many intrathoracic operations. The purpose is rather to point out some of the dangers that accompany this form of anesthesia in order that therapeutic measures may prevent a fatal termination. It has been shown that a very small increase in intrabronchial pressure may produce untoward results both during and following an operation. (In one case reported in our own clinic only 11 mm. of mercury pressure were used.)

Although very low pressures are usually employed in administering this type of anesthesia, a momentary obstruction to the escape of the gas within the lung raises the intrabronchial pressure to a high level, and little time is necessary in which to produce a laceration of the lung parenchyma. Much lower pressures are necessary to cause this injury

to the alveolar walls than to the visceral pleura. Our experiments as well as Macklin's substantiate Coryllos'[17] conclusions that an intrabronchial pressure of from 52 to 58 mm. of mercury was necessary to produce a rupture of the visceral pleura. In view of the above findings, re-expansion of the remaining lung tissue following intrathoracic operations should not be by marked distention with positive intrabronchial pressure, but by aspiration of air from the pleural cavity through a catheter or needle following its closure, care being exercised not to produce too much negative intrapleural pressure.

The above dangers are perhaps considerably greater when an intratracheal catheter is used. It is for this reason among others that, in our clinic, simple pressure face-mask anesthesia has been substituted, for the most part, for intratracheal catheter anesthesia in thoracic operations. It has been found that the operation is just as well tolerated by the patient and the condition following operation is as good or better with this type of administration. Our conclusions are based upon an experience in over fifty intrathoracic operations, including resection of the thoracic esophagus, removal of mediastinal tumors, repair of diaphragmatic hernias, and removal of lung tissue.[18] Probably the best indication for intratracheal anesthesia is in lobectomy for bronchiectasis where the cough is very productive and aspiration of exudate during operation may be necessary. In other cases simple pressure mask anesthesia has the added advantage in that there is no irritation of the tracheobronchial tree produced by the insertion of a catheter, and mouth organisms are not as apt to be carried into the distal passages.[19] This is especially true in all but pulmonary suppurative disease. It also does not entail the technical difficulties associated with the introduction of an intratracheal catheter which may be considerable in some cases. Anesthetists should, however, have immediately available and be skilled in the use of intratracheal anesthesia in case a sudden emergency should demand its use.

CONCLUSIONS

Positive pressure anesthesia has contributed much to the development and advancement of thoracic surgery.

Some hazards, such as mediastinal emphysema and spontaneous pneumothorax, associated with this type of administration have been little understood and appreciated.

High intrabronchial pressures for distention of the lungs are dangerous in intrathoracic operations and are unnecessary since a considerable reduction in functioning lung may be effected without materially interfering with the normal cardiorespiratory physiology.

Lung re-expansion following intrathoracic operations should be brought about chiefly by aspiration of air from the pleural cavity through a catheter or needle after closure of the thoracotomy instead of by positive pressure.

REFERENCES

1. Graham, E. A.: Some Fundamental Considerations in the Treatment of Empyema Thoracis, St. Louis, 1925, The C. V. Mosby Co.
2. Heuer, G. J., and Andrus, W. D. W.: The Alveolar and Blood Gas Changes Following Pneumectomy, Bull. Johns Hopkins Hosp. **33**: 130, 1922.
3. Adams, W. E.: Subtotal Atelectasis and Pneumonectomy in Dogs, Proc. Soc. Exp. Biol. & Med. **28**: 157, 1931.
4. Phillips, F. J., Adams, W. E., and Hrdina, Leo S.: Minimal Lung Function. In press.
5. Churchill, Edward D.: Segmental Pneumonectomy in Bronchiectasis, Ann. Surg. **109**: 481, 1939.
6. Ohsawa, T.: The Surgery of the Esophagus, Kyoto Imperial University, Tokyo, Japan.
7. Humphreys, George H., Moore, Richmond L., and Maier, Herbert C.: Studies of the Cardiac Output of Anesthetized Dogs During Continuous and Intermittent Inflation of the Lungs, J. THORACIC SURG. **7**: 438, 1938.
8. Magill, I. W.: Anesthesia in Thoracic Surgery With Special Reference to Lobectomy, Proc. Roy. Soc. Med. **29**: 7, 1936.
9. Stephens, H. Brodie: A Consideration of Contralateral Pneumothorax as a Complication of Intrathoracic Operations, J. THORACIC SURG. **5**: 471, 1936.
10. Bradshaw, H. H.: Anesthesia for Intrathoracic Operations, J. THORACIC SURG. **8**: 293, 1939.
11. Bourne, Wesley: Personal communication.
12. Heidrick, Fausteena, Adams, W. E., and Livingstone, H. L.: Spontaneous Pneumothorax Following Positive Pressure Intratracheal Anesthesia: Report of a Case. Arch. Surg. (In press.)
13. Elsberg, Charles A.: Further Experiences With Anesthesia by the Intratracheal Insufflation (Meltzer), With Remarks Upon the Method for Thoracic Surgery, Ann. Surg. **52**: 23, 1910.
14. Eisenbrey, A. B.: Observations on the Use of Intratracheal Anesthesia in Experimental Work, Surg. Gynec. & Obst. **15**: 715, 1912.
15. Macklin, C. C.: Pneumothorax with Massive Collapse From Experimental Local Over-Inflation of the Lung Substance, Canad. M. A. J. **36**: 414, 1937.
16. Marcotte, R. J., Phillips, F. J., Adams, W. E., and Livingstone, H.: Differential Intrabronchial Pressures and Mediastinal Emphysema, J. THORACIC SURG. **9**: 346, 1940.
17. Coryllos, Pol. N.: Mechanical Resuscitation in Advanced Forms of Asphyxia, Surg., Gynec., & Obst. **66**: 698, 1938.
18. Phillips, F. J., Livingstone, H. L., and Adams, W. E.: Anesthesia in Intrathoracic Surgery: Results in Over 50 Cases. In press.
19. Lewis, Ivor: Bilateral Lobectomy for Bronchiectasis, Brit. J. Surg. **24**: 362, 1936.

DISCUSSION

DR. ALFRED GOLDMAN, San Francisco, Calif.—About a year ago I did some experiments on dogs which confirmed those reported by Dr. Adams. That is, in dogs, mediastinal emphysema occurred when we placed needles in the side of the chest and introduced air under the skin at a pressure equal to 40 cm. of water. That death was concomitant with the occurrence of bilateral pneumothorax was proved by placing a cannula through the diaphragm and measuring the intrapleural pressure on a kymograph. When death occurred, there was a rise in the intrapleural pressure.

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—As I said, we have had no injuries of the lungs with the type of anesthesia we have used, but we have not gone up to pressures over 20 cm. of water. That all corresponds to about 20 or 30 mm. of mercury, I think. In going higher than that, there would be a risk and the lung might rupture.

DR. J. J. SINGER, Los Angeles, Calif.—The question of the expansion of pulmonary tissue, either by positive pressure or by the actual increase in size when

the lobe or lobes are removed, has been of great interest. Sometimes one sees the anesthetist use considerable pressure with his hand on the anesthesia bag, and undoubtedly some of the alveoli are ruptured. We have noted this in the study of pathologic specimens following the removal of the lobe. There was one dramatic case that Dr. Graham and I had a few years ago. This patient had a very large number of cysts in one lung. In fact, the lung was nothing but a mass of cysts ranging from pinhead size to the size of a grapefruit. When the lung was removed the patient was sent back to his room with a catheter in the pleural space. The end of the catheter was placed in fluid on the floor; aspiration from the chest cavity was produced that way. Within an hour or two we were called back suddenly to see the patient. He was in great distress, and we found that the opposite lung had encroached far over into the space left by the removal of the lung. In taking pleural pressure we found that such a high negative pressure existed in the pleural space that was operated upon that we decided to put fluid back into the space. He died the following morning, and the specimen showed that the entire remaining lung was so distended that it was functionless.

DR. SIDNEY SHIPMAN, San Francisco, Calif.—I would like to ask Dr. Adams a question about the exact mechanism involved in the production of the pneumothorax. Does this occur by extension of the mediastinal emphysema?

DR. W. E. ADAMS.—Time did not permit going into the details of the experimental work that we carried out. I only gave you a résumé of the most important findings, but we did test the effect of pressure on the anesthesia bag and other manipulations and it is surprising how much increased intrabronchial pressure results when one squeezes on the bag, as is often done. The intrabronchial pressure may rise as high as from 25 to 30 mm. of mercury. True, this is only momentarily, but it may be registered by a small catheter introduced into the bronchial tree and connected to a kymograph. I did not come here to criticize too severely positive pressure anesthesia or intratracheal anesthesia, but principally to stimulate interest in the dangers that accompany its use. We know that high intrabronchial pressures are necessary to break through the periphery of the lung, almost 60 mm. of mercury pressure, but as these experiments show, much less is needed to rupture the parenchymal tissue within the lung itself. I believe Dr. Macklin's theory regarding the development of mediastinal emphysema in these cases is correct—that the air follows along the blood vessels adjacent to the air passages and into the mediastinum and up into the neck, as subcutaneous emphysema. This would seem to be the best explanation of the production of mediastinal and subcutaneous emphysema in the referred case of asthma.

In regard to spontaneous pneumothorax, I believe that the same explanation may hold. I have observed total atelectasis of one lung accompanied on the same side by spontaneous pneumothorax in rabbits, goats, and dogs. In dogs the mediastinum permits air to go freely from one pleural cavity to another, but in the other two animals a stable mediastinum is present. Therefore, it should be much easier for the pneumothorax to develop by means of air breaking through the alveolar walls of the remaining overinflated lung, course down along the vessels to the mediastinum and thence into the pleural cavity on the side of massive atelectasis. The pneumothorax develops on the side of atelectasis because the intrapleural pressure is more negative on this side than it is on the side of the remaining overinflated lung tissue.

HEMICARDIAC HYPERTROPHY DUE TO INCREASED PERIPHERAL RESISTANCE

A Study of Pulmonic and Aortic Stenosis Experimentally Produced

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THAT cardiac enlargement as demonstrated clinically may be due to hypertrophy, to dilatation, or to a combination of both, is an obvious fact. Not so obvious are the factors responsible for the hypertrophy or the dilatation, whether dilatation occurs alone, whether it invariably precedes hypertrophy, or whether an increase in peripheral resistance alone may produce such hypertrophy through increased work. In previous studies[1] on pulmonic and aortic stenosis produced in adult animals certain information was obtained. A stenosis which permanently constricted the pulmonary artery to a circumference less than one-half the normal size was invariably fatal. Pulmonic stenosis of marked degree was immediately followed by an acceleration in pulse rate and by a drop in general blood pressure, both of which gradually approximated normal. The stenosis was followed immediately by a dilatation of the right ventricle, a dilatation which gradually subsided during the subsequent months until the heart again assumed a normal size as shown by roentgenographic studies. At necropsy a slight thickening of the right ventricular wall was found, and, microscopically, the right heart showed evidence of hypertrophy. There was no demonstrable increase in total blood volume after pulmonic stenosis. The production of the pulmonic stenosis resulted in a well-marked systolic bruit followed by an accentuated pulmonic second sound.

Aortic stenosis beyond the left subclavian artery lowered peripheral blood pressure but caused no acceleration in pulse. Such a stenosis not only lowered femoral pressure but increased carotid pressure. Well-marked aortic stenosis beyond the subclavian vessels of six months' duration produced no roentgenographic increase in the size of the heart; at necropsy there was no apparent dilatation of the heart, but there was a slight increase in thickness of the left ventricular wall, which showed a slight hypertrophy microscopically. The total blood volume remained relatively unchanged in the presence of an aortic stenosis. Partial constriction of the pulmonary artery or aorta by a metal band or tape frequently ended fatally through erosion of the vessel wall.

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It was apparent from these studies in the adult animal that stenoses compatible with life produced only moderate effects. When prompt

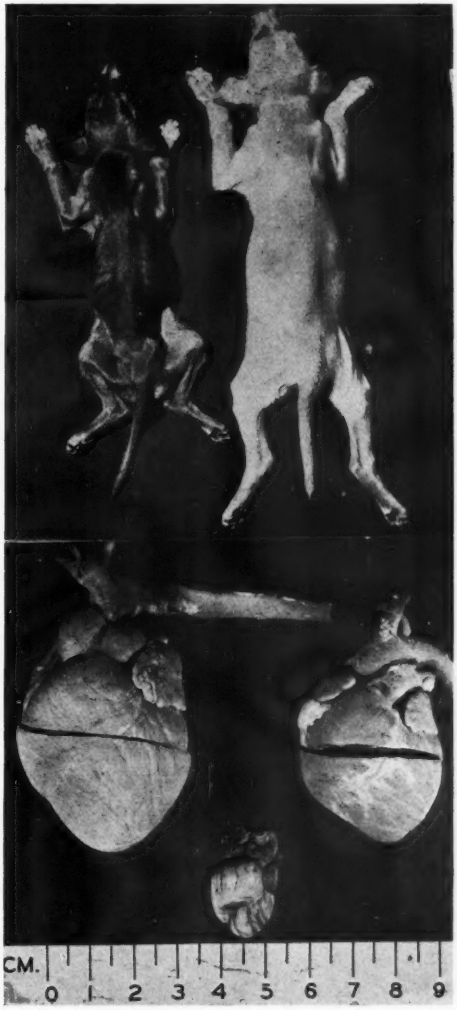

Fig. 1.—Experiment 1. Aortic stenosis in smaller animal of 44 days' duration. Note the much larger heart in the small experimental animal, as compared with the smaller heart in the much larger control animal. The very small heart represents the size of the heart of the animals at the time of the production of the stenosis.

and conspicuous effects were obtained, early death followed, either because of these effects or because of erosion of the vessel wall by the constricting ligature.

To obviate these difficulties in determining accurately the exact role in cardiac hypertrophy of peripheral resistance uncomplicated by distention of the cardiac chambers, experiments were attempted on newborn or very young puppies. In each instance, the heart of the young animal was exposed through a small anterior left intercostal incision, the pericardium opened, and a silk ligature placed around the root of either the pulmonary artery or aorta and tied so as to encircle the vessel snugly, but not to constrict it even slightly. It was thought that, as the animal and its organs grew in size, the vessel would remain constricted to its original size at the site of the ligature and a relative stenosis would occur. A litter mate was kept as the control animal. Many attempts ended fatally, but a few successes yielded instructive information.

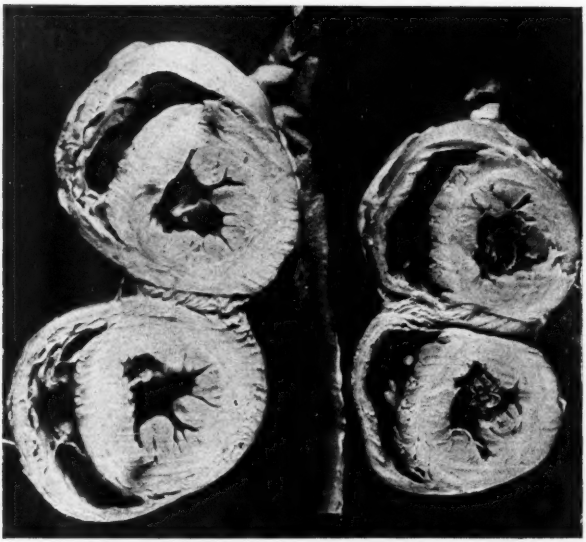

Fig. 2.—Experiment 1. Cross section of the large heart in the experimental animal with an aortic stenosis, compared with the cross section of the normal heart of a control litter mate. Note the greatly increased thickness of the left ventricular wall without dilatation of the central cavity.

EXPERIMENT 1.—Aortic stenosis. June 14, 1926. Under intratracheal ether anesthesia, the heart of a 72-hour-old pup was exposed, the pericardium opened, the aorta isolated as it left the heart, and a silk ligature applied.

On July 28, 1926, the operated animal was found dead. It had been slowly gaining weight and stature until four days before death, when it ceased eating, and it died of inanition. The operated animal was small and thin as compared with the control animal (Fig. 1) and weighed 1,245 Gm., the control weighing 1,650 Gm. Two hundred cubic centimeters of fluid were present in the pleural cavity and a similar amount in the abdominal cavity. The heart of the experimental animal weighed 29 gm., whereas the heart of the much larger control

animal weighed only 16.5 Gm. (Fig. 1). The left ventricle of the experimental animal was conspicuously thicker and firmer in all respects than that of the control animal, the former having an average thickness of 7 mm. and the latter an average thickness of 4 mm. The aorta at site of constriction admitted an 8 mm. bougie, whereas the aorta of the control animal admitted a 15 mm. bougie (Fig. 2).

EXPERIMENT 2.—Aortic stenosis. March 5, 1937. Under intratracheal anesthesia, through an intercostal incision, the heart of a 6-week-old puppy was exposed, the pericardium opened, the aorta isolated, and a ligature applied without constricting the artery. The operated animal weighed 2.7 kg., and the control, 1.8 kg. (Fig. 3).

Fig. 3.—Experiment 2. Photographs of pups preceding establishment of aortic stenosis in the darker animal on left, weight 2.7 kg. The control litter mate weighed 1.8 kg. The lower picture presents the pups a month before being killed, the experimental pup weighing 16.3 kg., the control pup, 14.8 kg.

On Jan. 14, 1938, roentgenograms revealed a cardiac enlargement in the operated animal (Fig. 4).

On March 16, 1938, both animals had grown to maturity, the operated animal weighing 16.3 kg., the control animal, 14.8 kg. (Fig. 3). The animals were anesthetized with chloroform, and the hearts were exposed and examined in situ. The heart of the control animal appeared normal in size and shape. The heart of the operated animal was larger, more vigorous in its action, and a marked thrill

could be felt in the ascending aorta. The heart of the operated animal weighed 178 Gm., that of the control animal, 130 Gm. On cross section, the left ventricle showed a remarkable increase in its musculature (Fig. 5), without any evidence of dilatation. The left ventricular wall had an average thickness of 10 mm. in the experimental animal as compared with 5 mm. in the control animal. At the site of the constriction, the aorta admitted a 20 mm. bougie; the artery proximal to it admitted a 40 mm. bougie; and the normal aorta of the control animal admitted a 34 mm. bougie.

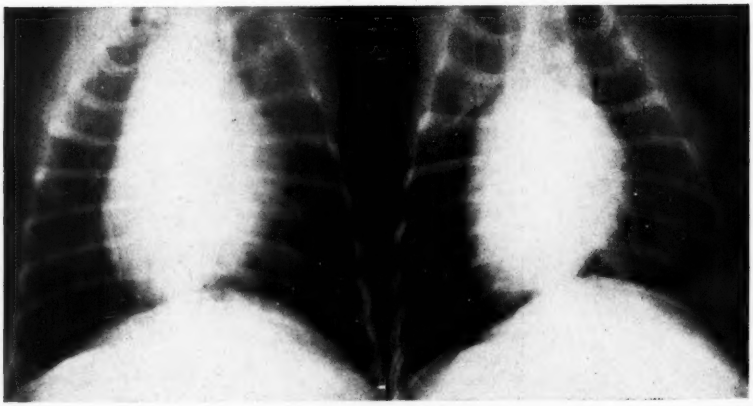

Fig. 4.—Experiment 2. Roentgenograms of chest showing increased cardiac shadow in animal having an aortic stenosis of 10 months' duration.

EXPERIMENT 3.—Aortic stenosis. On Nov. 24, 1938, the heart of a 12-week-old puppy was exposed through an intercostal incision, the pericardium opened, the aorta isolated, and a stout linen thread passed around it just beyond its emergence from the heart. This was tied without even slightly constricting the lumen. There was no dilatation of the ventricle. The animal made a good recovery and grew to maturity, as did the litter mate.

Electrocardiograms on May 15, 1939, yielded no difference in the experimental and control animals. Roentgenograms on July 17, 1939, showed a definite cardiac enlargement in the experimental animal (Fig. 6).

On Sept. 18, 1939, both animals were in good condition. In the experimental animal a loud systolic murmur was audible over the base of the heart, accompanied by an accentuated second sound. The total blood volume of the experimental animal, which weighed 8.6 kg., was 880 c.c., and that of the control animal, which weighed 7.1 kg., was 750 c.c. On Sept. 21, 1939, the two animals were killed. The heart of the experimental animal was large and weighed 97.5 Gm., whereas the heart of the control animal weighed 72.5 Gm. (Fig. 7). This represents a 34.4 per cent preponderance in the cardiac weight of the experimental animal, as compared with only a 21 per cent preponderance in total body weight. The aorta of the experimental animal admitted a 15 mm. bougie, whereas the aorta of the control animal admitted a 24 mm. bougie. The average thickness of the left ventricular wall measured 9 mm. in the experimental animal and 6 mm. in the control animal.

EXPERIMENT 4.—Pulmonic stenosis. March 25, 1931. Two small pups, 5 weeks old, each weighing 2.5 kg., were available for the experiment, one to be kept as a control. In attempting to isolate the pulmonary artery in the first animal, the wall was pierced, and the animal died on the table. Accordingly, the control was

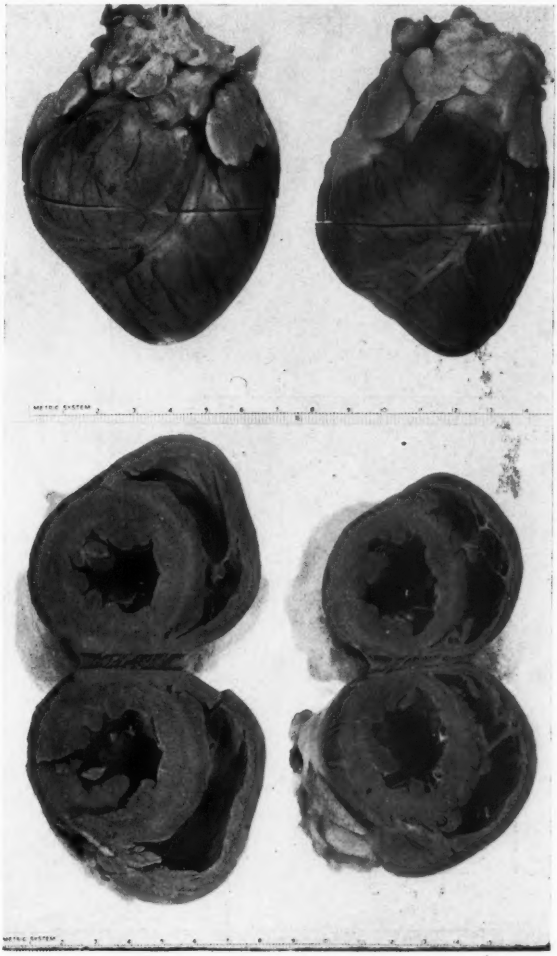

Fig. 5.—Experiment 2. The heart on the left (weight 178 Gm.) is that of an experimental animal having an aortic stenosis of 12½ months' duration. The heart on the right (weight 130 Gm.) is that of a control litter mate. Note the greatly increased thickness of left ventricular wall and the absence of cavity dilatation.

operated upon, and a doubled silk ligature applied to the pulmonary artery without constricting the lumen. The animal survived, grew well, and was killed Jan. 12, 1933. The heart showed a remarkable hypertrophy, limited to the right ventricle, the wall of which appeared almost as thick as the left ventricular wall (Fig. 8). The right auricle was not dilated, nor were its walls thickened.

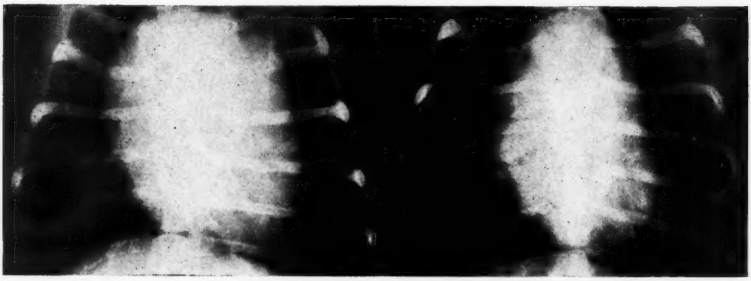

Fig. 6.—Experiment 3. Roentgenogram of chest showing cardiac enlargement in presence of aortic stenosis of 10 months' duration.

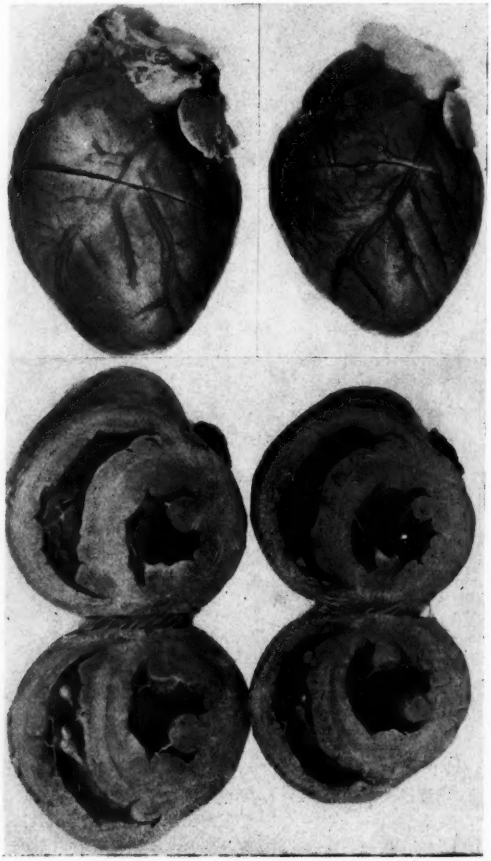

Fig. 7.—Experiment 3. The enlargement of the heart and hypertrophy of the left ventricle on the right followed an aortic stenosis of 10 months' duration. Note preponderance of left ventricle in heart on upper right.

EXPERIMENT 5.—Pulmonic stenosis. April 30, 1931. Two female collie pups, 6 weeks old, were operated upon under intratracheal ether anesthesia. The heart of the heavier animal (7.8 kg.) was exposed through an intercostal incision, the pericardium opened, and the pulmonary artery isolated. The artery, which had a diameter of 1 cm., was constricted by a ligature, diminishing its caliber by about one-third. The control animal weighed 5.2 kg.

Fig. 8.—Experiment 4. Heart of animal having a pulmonic stenosis of 22 months' duration. No control animal available. Note great thickness of right ventricular wall without dilatation of the cavity.

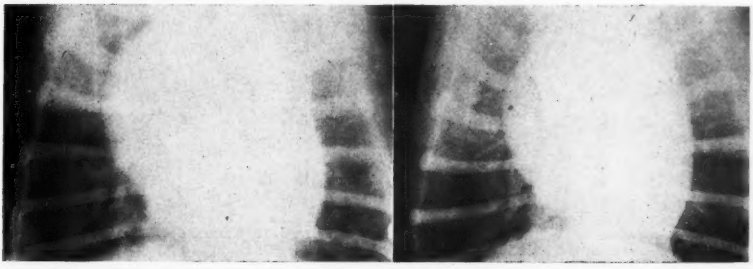

Fig. 9.—Experiment 5. Roentgenograms illustrating marked cardiac enlargement in presence of a pulmonic stenosis of 17 months' duration, as compared with the heart (on left) of a normal control litter mate.

By Sept. 30, 1932, both the operated and control animals had grown well, the operated animal being somewhat larger than the control. There was a very loud systolic murmur to be heard over the entire chest and out into the peripheral lung fields. There was no audible second sound. Roentgenograms revealed a marked cardiac enlargement in the operated animal (Fig. 9).

On Dec. 2, 1932, both animals, in excellent condition and fully grown (Fig. 10), were killed by chloroform. The heart of the operated animal was conspicuously

larger (Fig. 11) than that of the control, and the right ventricle and auricle were more prominent and obviously thicker than in the control. The heart of the operated animal weighed 220 Gm., and that of the control animal, 144 Gm. On cross section, the right and left ventricular walls of the operated animal appeared almost equal in thickness. By actual measurement, the right wall was 8 mm. thick, the left 10 mm. thick. In the control animal, the right and left ventricular walls measured 5 mm. and 10 mm., respectively. At the point of constriction the pulmonary artery admitted a 20 mm. bougie, whereas the pulmonary artery of the control animal admitted a 28 mm. bougie. The aorta of the operated animal admitted a 28 mm. bougie, and the aorta of the control animal admitted a 28 mm. bougie.

Fig. 10.—Experiment 5. Fully grown animals, the left animal having a pulmonic stenosis of 25 months' duration, the animal on the right being a control litter mate.

COMMENT

These studies yield certain instructive observations. Cardiac hypertrophy, limited to one-half the heart, was produced by increasing peripheral resistance alone, not accompanied nor preceded by dilatation. This hypertrophy occurred on either side of the heart, depending upon the site of constriction. It cannot, therefore, in the case of pulmonic stenosis be dependent upon an increased coronary flow such as might be invoked as a cause of the left ventricular response to a constriction

beyond the coronary arteries. This hypertrophy was sufficient to produce conspicuous cardiac enlargement, an enlargement which was quite different from that which followed the establishment of an arteriovenous fistula, either between peripheral vessels[2] or between the ventricles. In these latter two instances, there is a great decrease in peripheral resistance in the fistulous circuit. In the case of the peripheral fistula, as a

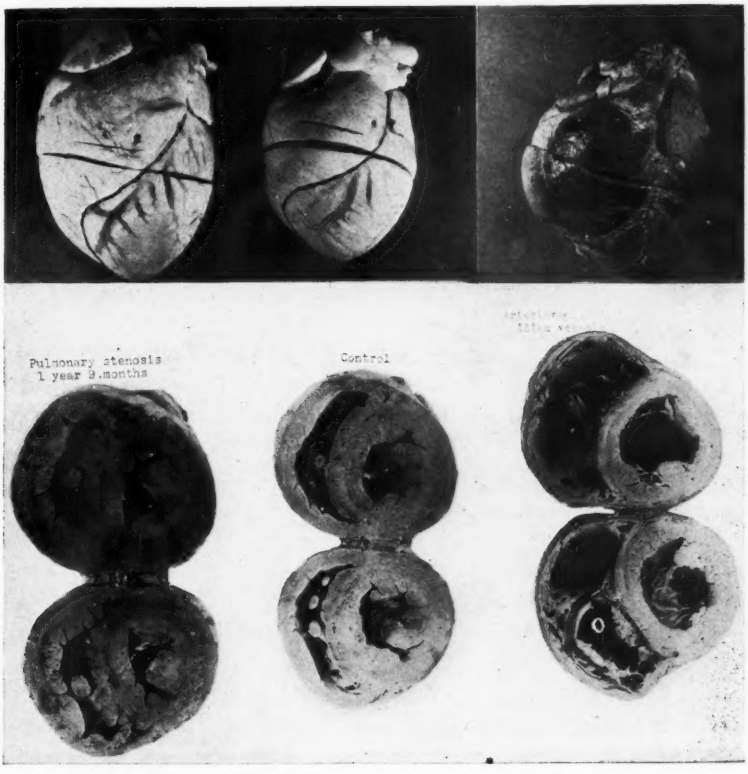

Fig. 11.—Experiment 5. Heart on left is that of an experimental animal having a pulmonic stenosis of 19 months' duration. Note cardiac enlargement in comparison with the heart of a normal control litter mate and markedly increased thickness of right ventricular wall, without dilatation of right ventricular cavity. Heart on extreme right is that of an animal having a right iliac arteriovenous fistula of 8 months' duration, illustrating marked cardiac enlargement due entirely to dilatation of ventricles with a corresponding thinning of the ventricular walls.

result of this decrease in peripheral resistance, there occurs a short-circuiting of a large volume of blood directly back to the heart, a doubling of the cardiac output, a great increase in the bulk of blood flowing through the shorter circuit, and a marked cardiac enlargement (Fig. 11). The extent of the cardiac enlargement was found to be determined by the size of the fistula or by the size of the interventricular

defect. The size also determined the volume of short-circuited blood and the increase in cardiac output. Incident to the deflection of this considerable volume of blood into the shorter circuit, there occurred a fall in general blood pressure which was commensurate also with the size of the fistula; this was gradually compensated by a corresponding increase in the total blood volume, an increase which was demonstrated in the presence of both the peripheral and the intracardiac fistulas. Many of the interesting characteristics of the peripheral fistula are dependent upon this increase in total blood volume.

It is further apparent from these studies that cardiac enlargement may be due to two very different factors: In pulmonic and aortic stenosis, the enlargement is due to hypertrophy, limited to one-half of the heart. This hypertrophy is due to the increased peripheral resistance against which the cardiac musculature must work. In the presence of the peripheral fistula, the enlargement is almost entirely due to dilatation with thinning of both ventricular walls. In this instance there is a decrease in peripheral resistance, but an increase in the volume or bulk of blood flowing through the heart.

The term "cardiac hypertrophy" is often loosely applied to any enlarged heart demonstrated clinically by roentgenogram or by physical signs. The term "cardiac enlargement" is obviously more accurate and is the preferable term when describing large hearts observed clinically, leaving in abeyance the question of hypertrophy or dilatation until confirmation of either at the necropsy table or at the operating table.

Operations upon the heart and its large vessels are becoming increasingly more frequent, and one may prophesy that it will become increasingly important, as the range of these operations is extended, to be able to recognize whether the enlargement of the heart or of its individual chambers is due to dilatation or to hypertrophy. The operation to be performed, if any, may depend upon the correct interpretation or differentiation of these two conditions at the operating table.

SUMMARY

Aortic stenosis was produced in three very young animals, resulting in marked hypertrophy of the left ventricle without dilatation of the ventricular cavity.

Pulmonic stenosis was produced in two very young animals, resulting in marked hypertrophy of the right ventricle without dilatation of the ventricular cavity.

Hemicardiac hypertrophy under these two conditions is due to an increased peripheral resistance, unaccompanied by ventricular dilatation. This is in marked contrast to the cardiac enlargement seen in the presence of a large arteriovenous fistula, which is preponderantly due to a

dilatation and only in slightest degree to an hypertrophy. In the presence of a fistula there are a decrease in peripheral resistance at the site of the fistula and an increase in cardiac output, the extent of these changes being dependent upon the size of the fistula.

Cardiac enlargement observed clinically or radiographically, therefore, may be due either to dilatation or to hypertrophy. It is inaccurate to refer to such enlargement as "cardiac hypertrophy" until proved by direct observation of the heart.

REFERENCES

1. Holman, Emile, and Beck, Claude S.: The Physiological Response of the Circulatory System to Experimental Alterations. III. The Effect of Aortic and Pulmonic Stenoses, J. Clin. Investigation 3: 283, 1926.
2. Holman, Emile: Arteriovenous Aneurysm. Abnormal Communications Between the Arterial and Venous Circulations, New York, 1937, The Macmillan Co.

THE POSTNATAL GROWTH OF THE LUNG

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THE more important evidence concerning the character of the postnatal growth of the lung has been histological. The presence of tubular sprouts and mitoses without evidence of distention of the lungs is the most significant detail of the microscopic picture. Bremer,[1] who has made careful histological studies in several species, has found these features regularly present in the growing animal. He points out that it is the presence of the ability for growth in the adult rat as contrasted to the lack of this ability in the adult dog which accounts for the difference of opinion in regard to regeneration as expressed by Hilber[2] and Rienhoff, Reichert, and Heuer.[3] Rienhoff and co-workers made no quantitative estimation of regeneration on their material, simply stating that from microscopic section the lung appeared dilated. Dawson[4] found that, in the rat, ossification and union of the heads of the ribs were only beginning in animals 30 months of age. In all old rats examined, cartilage plates were observed in both ribs and sternum; hence, skeletal growth may continue throughout the life span. Bremer[5] observed that this would permit continued growth of the pleural cavity to accommodate the lungs. It is conceivable that the fixation of the size of the thoracic cage due to the cessation of skeletal growth, and not the age of the animal per se, is the important factor in determining whether or not the lung will regenerate. The age of the animal simply determines the speed of the restitution. Donaldson[6] showed on the Wistar strain of rats that the lung weight increased with age and was always proportionate to the increasing body weight. Cohn[7] confirmed the constant relationship of the lung weight to body weight on the strain of rats with which he was working. He then demonstrated that, when a large amount of lung tissue is removed, the remaining lung tissue will increase in weight until it equals the predicted normal lung weight for a rat of a particular body weight. The speed of this restoration of weight was very great in the young rats, gradually becoming slower as the rats aged. He then showed that, if the pull of the chest wall was eliminated by any one of various collapsing methods, the lung weight fell below the predicted body weight for the rat. Lowering the atmospheric pressure and thus increasing the depth of respiration caused the lung weight to increase until it was higher than the predicted weight. It was concluded, therefore,

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that the growth of the lung followed the growth of the thorax in a purely mechanical fashion, rather than vice versa. There has been some evidence from tissue culture methods that tension on fibroblasts increased their rate of growth.[8] That the increase in weight which Cohn found was actually a protein increase was demonstrated by Addis[9] who showed that the nitrogen content of the lung paralleled the weight change.

In order to determine whether or not the increased weight of the lung of the rat represented an actual increase in number of alveoli, an approximate method of counting alveoli has been devised, and the results are now recorded.

METHODS

Rats were kept under standard conditions of housing and feeding. When an experiment was completed, the animal was anesthetized with ether, the abdomen opened, and the aorta cut. After bleeding had stopped, the thoracic viscera were removed and trimmed. A cannula was then tied into the main bronchus of the left upper lobe, and Bouin's fluid was forced in. A U-tube in series with the injection apparatus allowed the pressure to be no higher than 2 cm. of mercury. After the lobe had been completely distended, the bronchus was tied and the lobe placed in Bouin's solution for twenty-four hours. The lobe was then dehydrated with dioxane solutions.

Before the lobe was placed in paraffin, its volume was measured by the amount of solution it displaced in a calibrated cylinder.

Paraffin sections were then made and stained. Using high power, several evenly distended fields were counted. Fields containing large vessels or bronchi were avoided. All alveoli or parts of alveoli in each field were counted, and the average of three high power fields was used in calculating the number of alveoli present in the left upper lobe.

The calculation was based upon the assumption that alveoli are polyhedral, approaching spheres, and that the cross sections approach circles, so that the area of the field counted, divided by the number of alveoli, gives the average of an infinite number of cross sections through spherical alveoli. The average cross section through a sphere has the same area as that of a cylinder as high as the diameter of the sphere and of the same volume as the sphere. The average volume of the alveoli counted was calculated on the assumption that the area of the microscopic field, divided by the alveolar count, gave the average area of a cross section of a cylinder equal in height to the diameter of the alveoli.

$\frac{4}{3}\pi x^3$ = volume of a sphere of radius x. A cylinder of height x, and of the same volume as the sphere, will have a cross section area a equal to the average cross section area of an infinite series of haphazard sections through the sphere. In these experiments, a was determined by dividing the area of the high power field counted, 125,000 mu^2 for a field 0.4 mm. in diameter, by the average alveolar count, c.

Then, $4.19\ x^3 = 2(\frac{125{,}000}{c})$, and $x^2 = \frac{59{,}600}{c}$. The average alveolar volume equals $4.19\ x^3$. Dividing the volume of the lobe by the average alveolar volume gives the number of alveoli in the lobe.

In Table I are given the alveolar counts on left upper lobes in a series of normal animals and in a series of animals subjected to procedures which either increased or decreased the weight of the left upper lobe as compared to normal controls.

While the method of counting is only an approximate one, an attempt was made to adhere as closely as possible to the same routine so that the observations would be comparable. The observations would indicate that the increase in weight was due in part to an increase in the number of alveoli, and in part to an increase in the thickness of the alveolar wall. Certainly the time factor was important, as well as the age of the animal. The lowered counts immediately after pneumonectomy suggest that, with the immediate increase of tension on the remaining lung, the alveoli are stretched and broken first, and then new alveoli begin to grow, their rate depending upon the age of the rat. This explanation would be in agreement with Hilber and Bremer.

TABLE I

ALVEOLAR COUNTS OF LEFT UPPER LOBES

PROCEDURE	NUMBER OF RATS	AVERAGE BODY WEIGHT (GM.)	AVERAGE NUMBER OF ALVEOLI IN LEFT UPPER LOBE
Normal rats	7	79	
	7	168	
	15	256	4,910,000
	10	330	
	6	389	
Removal of 66 per cent of lung tissue:			
3 days postoperative	8	108	3,910,000
	5	121	3,420,000
	5	198	3,590,000
7 days postoperative	5	90	5,280,000
	5	196	4,520,000
30 days postoperative	17	142	6,900,000
	5	231	5,310,000
Low atmospheric pressure (350 mm. of mercury)	6	37	7,010,000
	6	195	6,560,000
Left phrenicectomy	8	126	5,020,000

The decrease in the lung to body weight proportion following phrenicectomy could not be demonstrated to be due to a decrease in the number of alveoli.

SUMMARY

Following removal of lung tissue in the rat, the increase in size of the remaining lung is accompanied by a new growth of alveoli.

An initial decrease in alveoli is evident three days after pneumonectomy, return to normal number within one week, and an increase above normal, even in older rats, at the end of thirty days.

Compensatory hypertrophy and formation of new alveoli occur more rapidly in young rats than in old rats.

REFERENCES

1. Bremer, J. L.: Postnatal Development of Alveoli in the Mammalian Lung, Carnegie Contrib. to Embryol. **25**: 83, 1935.

2. Hilber, H.: Experimenteller Nachweiss des formation Einflusses der Atemluft auf regenerierende Rattenlungen, Morphol. Jahrb. **74**: 171, 1934.
3. Rienhoff, W., Reichert, F., and Heuer, G.: Compensatory Changes in the Remaining Lung, Following Total Pneumonectomy, Bull. Johns Hopkins Hosp. **57**: 373, 1935.
4. Dawson, A. B.: Further Studies on the Epiphyses of the Albino Rat Skeleton, Anat. Rec. **34**: 351, 1927.
5. Bremer, J. L.: The Fate of the Remaining Lung Tissue After Lobectomy or Pneumonectomy, J. THORACIC SURG. **6**: 336, 1936-37.
6. Donaldson, H. H.: The Rat, Ed. 2, Memoirs of the Wistar Institute, No. 6, Philadelphia, 1924.
7. Cohn, R.: Factors Affecting the Post Natal Growth of the Lung, Anat. Rec. **75**: 195, 1939.
8. Stearns, M. L.: Growth and Differentiation of Connective Tissue as Observed Microscopically in the Living Rabbit (Abst.), Am. J. Med. Sc. **198**: 144, 1939.
9. Addis, T.: Compensatory Hypertrophy of the Lung Following Unilateral Pneumonectomy, J. Exper. Med. **47**: 51, 1928.

NOTES ON GUNSHOT WOUNDS OF THE CHEST

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HISTORY

THE inclusion of the term gunshot in the title implies that the wound has been caused by some missile propelled by an explosive or having explosive properties or with a combination of these two. There is much difference of opinion as to when gunpowder was introduced, but it appears that it did not become really effective until the end of the fifteenth century. Up till this time wounds were caused by more primitive weapons, such as swords, arrows, or daggers, and the accounts of treatment show that this, like the weapons, was primitive. It was at one time believed that suppuration was necessary for proper healing, and if suppuration did not occur, various suppurative medicines were employed. Mondeville, in the fourteenth century, is believed to have been the first to demonstrate that suppuration was not necessary in the healing of wounds. For penetrating wounds of the chest he advised removal of foreign bodies and immediate closure. When the use of gunpowder became more common, more severe wounds were the rule. It was thought by many that gunpowder caused poisoning of the flesh, and in order to neutralize this effect, it was the practice to cauterize the wounds by injecting boiling oil. Ambroise Paré found that, when he ran short of oil and could no longer cauterize the wounds, they did as well or better without it.

There does not seem to have been any important advance in the treatment of chest wounds until the early part of the nineteenth century. John Hunter saw service as an army surgeon with the expedition to Belleisle in 1761 and has put down many astute observations, but he has added little to the subject in the way of improvements in treatment. His account of gunshot wounds of the chest opens with the following statements: "Little notice has been taken of wounds of the chest and lungs; indeed it would appear at first sight that little or nothing could be done. Yet in many cases a great deal may be done for the good of the patient." He recognized the fact that all wounds of the chest are not mortal and also appreciated some of the factors which make chest wounds different from those in other parts of the body; the chief of these, according to him, is the collapse of the lung which takes place when a wound is made into the chest. This results in a disproportion between the chest and its contained viscera; it prevents healing by first intention, for the space must become filled with air or blood or both and, therefore, adhesion cannot readily take place. He mentioned the possibility of treating a hemothorax by allowing the fluid to run out of the wound. This suggestion has been recently revived and will be commented upon later. For a large hemothorax he advised the "operation for the empyema." He recognized the fact that the collapse of the lung is not an invariable consequence of wounds of the lung and that the collapse might be prevented by adhesions of lung to chest wall. The account does not furnish any statistics, and, as far as I can find, there are no reliable figures available before the early part of the eighteenth century.

Both Guthrie of the British service and Baron Larrey of the French service were responsible for the introduction and advocacy of improved methods of treatment. It was Guthrie's practice to clean the wound, remove the missile if accessible, and close the wound by suture. Baron Larrey's practice was very similar; he

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advised excision of the wound, removal of loose splinters of bone, removal of the missile if within reach, and then closure of the wound with plaster.

Dr. Patrick Fraser has written an exhaustive account of chest wounds, based upon his experience in the Crimean War. At this time venesection was the common method of averting internal hemorrhage and of combating inflammation, and Fraser was a strong opponent of this treatment. The views which he expressed on this subject aroused a great controversy among British military surgeons of the period. Speaking of this practice of what he calls "bleeding the bled," Denys, in *The Cloister and the Hearth*, says that if a soldier has the good luck to be wounded where no doctor can be had he will live—a not very flattering commentary on surgical methods.

Otis gives a clear and excellent account of chest wounds in the American Civil War. Several wounds of the heart are described, but no account is given of any attempt to suture the heart itself. It is remarked that wounds of the heart are not necessarily fatal. There are instructive remarks on the treatment of retained foreign bodies, and the advice given is a middle path between rash interference and timid and harmful noninterference. The conclusion is that retained foreign bodies are more likely to cause serious mischief than to become encapsulated and remain innocuous. Regarding pneumothorax, one is surprised to read the following: "It was the general experience during the war that traumatic pneumothorax very rarely assumed such a phase as to excite alarm. In the vast series of cases of chest wounds this complication is noted as troublesome in less than half a dozen." In that connection, however, it should be recalled that most of the chest wounds in that war were caused by small missiles. Various treatments for hemothorax are discussed, and Chassaignac is quoted as having suggested the injection of air into the pleural cavity in order to encourage collapse of the lung and cessation of the bleeding. There is a long discussion on the advisability or otherwise of bleeding as a routine treatment for chest wounds, and the conclusion that it is better not to bleed is arrived at.

The experiences and writing of surgeons in the Franco-Prussian War do not appear to have added anything of value to the treatment.

Mortality figures will be given later, but here it may be noted that penetrating wounds of the chest were, until the Russo-Japanese and the South African Wars, regarded as of very grave, if not fatal, import. In both these last-mentioned wars the majority of wounds were inflicted by small caliber rifle bullets, frequently at long range. The general principle of treatment was expectant, the wounds were often mere punctures, and the only operations performed were for empyema. In South Africa the climate was good, the soil dry and free from contamination, and there the mortailty for chest wounds was the lowest on record. Elliott and Gask quote Sir George Makins as saying "there is no reason why a perforation by a bullet of small caliber should be more feared than a puncture from an exploratory trocar, and the danger of the two wounds is probably very much the same." During the whole campaign, Makins is said to have seen only one case of empyema. Aspiration was regarded as not only useless, but harmful, and was condemned; resection of rib to dislodge intrapleural blood clot was also forbidden—and rightly. It may be said then that in South Africa chest wounds were left to nature and that they did extremely well. Many people thought that this reduction in mortality was due to the introduction of the antiseptic method into surgery, but really it was due to a different type of wounds and excellent climatic conditions.

At the outbreak of the Great War in 1914, surgeons were mostly of the opinion that chest wounds required little in the way of active treatment. But conditions now were vastly different. The more modern weapons employed produced very extensive tearing wounds, and the soil was such that gross contamination was the

rule. Hemorrhage and sepsis accounted for a very considerable part of the high death rate. Then came the more extensive use of aspiration for both diagnostic and therapeutic purposes, and then the drainage of the pleural cavity when it proved to be infected, but even these measures did not produce any marked reduction in mortality.

In order to reduce the high mortality from hemorrhage and sepsis, Duval and others, in 1916, commenced to do radical operations, and these were found to be quite practicable but did not apparently, in average hands, result in any marked improvements in results. It was not until better selection of cases for operation became the rule that any good results were obtained. The medical services at the end of the Great War were very highly organized, and this permitted of teamwork in the treatment of chest wounds. For the best results there had to be cooperation between physician, surgeon, bacteriologist, and radiologist and careful selection of cases requiring operation. Details of the nonoperative and operative treatment in vogue at the end of the Great War may be found in the writings of the various authorities quoted.

INCIDENCE AND MORTALITY

In the figures given by various writers on past wars there is a remarkable uniformity in the proportion of chest wounds to all others, and it seems that about one wound in every twelve is of the chest. There are many obvious difficulties in assessing the number who die in the field of chest wounds, but to say that it is from one-third to one-half of the total killed is probably near the truth. When we come to study the number of deaths among the wounded brought in for treatment, we find an extraordinary difference in statistics. There is nothing to be gained by quoting a long list of figures, but the following small list is instructive. These figures refer to penetrating wounds.

English in the Crimea	79.2%
American Civil War	62.6%
Franco-Prussian War (Germans)	56.7%
Spanish-American War (U. S. Troops)	24.5%
Boer War (British)	14.0%
Great War (British)	27.5%
Sino-Japanese War, 1937 (Chinese)	14.8%

Thoracic surgery is a subject which has been making tremendous strides in the last ten to twenty years, and one would expect that similar strides would be made in the treatment of chest wounds, but this does not appear to be the case. I have been unable to find any accounts of war surgery in Spain or Abyssinia, but gather that no momentous contributions have been made to the treatment of chest wounds in either of these campaigns.

An opportunity for the study of chest wounds was afforded me during the hostilities which took place around Shanghai in 1937. It is regrettable that it was impossible to take full advantage of the opportunity, and in many cases it was very difficult to make a full

study or to keep detailed notes, mainly owing to war conditions. Full and accurate statistics could not be kept.

In the Shanghai hostilities the method of treatment employed did not differ greatly from that advocated by the British authorities at the end of the Great War except that radical surgery was not often employed.

This paper consists of notes and comments upon chest wounds in general and upon 150 cases observed and treated here. It cannot be claimed that anything original or revolutionary in the way of treatment has been introduced, but the paper may be of interest to those called upon to treat such injuries.

IMPORTANT POINTS IN THE APPLIED PHYSIOLOGY OF THE CHEST

Only those factors which have a bearing on the treatment of chest wounds are discussed, and by far the most important consideration is the effects of a pneumothorax. The dangers of an open pneumothorax have been recognized for years, and various explanations of the symptoms have been offered. The older explanations dealt largely with the question of collapse of the lung on the affected side and the consequent loss of breathing surface. Graham and Bell showed that there was also a diminution of the contralateral lung, but even this does not account for the severity of the symptoms. Today cases of bilateral artificial pneumothorax are common, and patients can stand a surprising amount of bilateral collapse without feeling any distress. Experimentally one lung has been completely put out of commission by ligature of the lung root without producing the symptoms of open pneumothorax. Other explanations have been brought forward, such as partial anoxemia due to the blood which passes through the affected lung remaining unoxygenated and the "Pendelluft" phenomenon of Brauer. By the latter is meant the seesaw of air which takes place between the lungs across the bifurcation of the trachea, resulting in an unprofitable exchange of air between the lung of the sound side and the lung of the wounded side and thus having the obvious result of limiting alveolar space.

There can be little doubt that the factor of greatest importance is the mediastinal flutter and its deleterious effects upon the great veins bringing blood to the heart. Systolic output is dependent upon diastolic inflow and the mediastinal flutter causes periodic obstruction to both superior and inferior venae cavae and consequent serious circulatory phenomena. Where the mediastinum is fixed by disease, it is usually quite safe to make an open pneumothorax, for, although the lung collapses, there is no question of mediastinal flutter. The symptoms of open pneumothorax are, therefore, due to circulatory disturbance rather than to the respiratory disturbance per se.

Other important points about open pneumothorax are the rapid loss of heat from the chest and the great tendency to infection. The size of the opening in the chest wall which is compatible with life is of importance and bears a direct relationship to the vital capacity of the patient, as was shown by Graham and Bell. It has been found that at operation quite large openings can be made, provided the mediastinum can be prevented from flapping by some simple means, such as grasping and pulling the lung into the wound.

Tension pneumothorax produces even more marked symptoms of distress than the open variety, for the pressure in the pleural cavity of both sides approaches a positive value. The condition on the right side produces its ill effects more rapidly than when it is on the left side, for the right heart and great veins are more sensitive to pressure than the left heart and main arterial trunks. A tension pneumothorax, unless relieved, invariably leads to a fatal termination.

Effect of Blood in the Pleural Cavity.—The source of the blood will be discussed later. A collection of blood in the pleural cavity will, of course, cause collapse of the lung to a degree depending upon the amount of fluid. The pressure of the pulmonary arteries is low, about one-fourth of that in the systemic vessels, and so there is a tendency towards spontaneous arrest of bleeding from the lung when a balance is obtained between collapse of the lung and pressure in the pleural cavity.

Removal of large quantities of fluid from the chest cavity is frequently found to cause unpleasant and dangerous cardiac and respiratory symptoms which can be counteracted by air replacement.

Cardiac Tamponade.—The phenomenon of cardiac tamponade has been known for a very long time. There is normally a small quantity of fluid in the pericardial sac, but once the amount exceeds 200 c.c., pressure is exerted on the heart, and the more rapid the production of this pressure, the greater the deleterious effect. The thicker-walled ventricles are not so adversely affected as are the auricles and great veins. The heart can be handled for operative purposes, provided the handling is gently and gingerly performed and frequent stops are made to allow the heart to adapt itself to the unaccustomed conditions. Safe intervals for compression of the various parts of the heart are given as follows: venous pedicle for from one-half to one minute; coronary circulation for two minutes; vena cava for ten minutes; and pulmonary artery and aorta for from one to two minutes.

CLINICAL ASPECTS

Practically all our cases were of the penetrating variety, and the wounds varied from small and almost immediately sealed bullet holes to large and gaping openings in the chest wall.

Of thoracoabdominal wounds we saw very few; probably most of them died before reaching the base hospitals. A great many of the wounds were of the through-and-through variety; in these the exit wound was sometimes larger than the entrance, sometimes the same size, and sometimes even smaller. A certain number had retained projectiles in the chest, and in some instances the projectile had traversed the chest and stopped in the spine or abdomen.

EFFECTS OF WOUNDS

1. Shock and collapse. The majority of the more extensive wounds showed a marked degree of collapse.

2. Pneumothorax, open or closed, was present in nearly all cases. In the larger wounds, air could usually be heard entering and escaping. In two cases tension pneumothorax was observed.

3. Hemothorax was almost invariably present.

4. Pyothorax was a very common sequel, and frequently all three, pneumothorax, hemothorax, and pyothorax were combined. The pyothorax usually took the form of empyema, but several cases developed lung abscess of the putrid variety.

5. Emphysema of varying degrees was seen. One man had such a marked case that both his eyes were closed, his lips were so swollen that he could scarcely eat, and his scrotum was the size of a football. One observer remarked that his appearance was suggestive of a gigantic frog which was a very apt description.

6. Brachial plexus injuries. There were two cases of extensive injury to the brachial plexus, and in one an axillary aneurysm developed.

7. Remote complications. Septicemia and pyemia sometimes occurred, and at least one man died of cerebral abscess. A good many of the patients developed dysentery, and this complication was responsible for some deaths. Many of the patients suffered from malnutrition, and beriberi was frequently observed.

EXAMINATION OF THE PATIENTS

When patients were first admitted, and especially during rush periods, the examination was often perfunctory, but an attempt was made to note the site and size of the entrance wound, and the site and size of the exit wound if present, and also whether or not the wound was of the "sucking" variety. In the latter case instructions were issued that the wound was to be covered at once by a large wet dressing until further measures could be adopted. Ordinary clinical methods of examination were employed, and special stress was laid on the degree of shock or collapse present, the amount of external hemorrhage, the position of the apex beat and the trachea, and the presence of dullness. Auscultation was not much employed. Hemoptysis is, of course, a very common symptom. This would suffice for the imme-

diate examination. Often injuries of other parts of the body were present, and these and the chest wound would then be attended to. Further examinations were usually required at a later date.

Effusions, whether blood or pus, were withdrawn when indicated and examined by the naked eye, the microscope, and sometimes cultured. Fluoroscopy and, in certain cases, the taking of x-ray pictures were largely employed.

Cases were examined as required, and once a week a séance was held when all patients with chest injuries were fluoroscoped and notes made of the findings. In empyema cases a chart was kept showing the size of the cavity from day to day. For this estimation the simple method was used of emptying the cavity and then finding out how much saline was required to fill it up. This method should be simple but requires attention to a few elementary principles, and it is surprising to see how badly it is often carried out. It is really better done with the patient on an operating table so that the head can be lowered at will and so that the body does not sag down in the middle as it is likely to do in bed. The patient must be put in such a position for the estimation that the opening into the cavity is at a higher level than any other part of the cavity. This is a more accurate method of estimating the size of the cavity than is fluoroscopy. In the presence of a bronchial fistula the method cannot be used.

All patients with cavities were examined with the thoracoscope, and although nothing striking was discovered, it was felt that a correlation of all the findings, clinical, radiologic, thoracoscopic, and operative, gave one a much clearer mental picture of the pathologic processes than if any of the methods had been omitted. The thoracoscope was used on six occasions in an attempt to visualize foreign bodies in the pleural cavity, but usually with no success. In one patient a shell fragment lying on the diaphragm was visualized with the thoracoscope and with its aid extracted with a long pair of forceps passed through the wound. In another an opening was seen in the diaphragm; through this opening a bullet had passed into the abdominal cavity.

TREATMENT

Many patients were admitted badly shocked and often bleeding externally or internally. After a rapid examination and estimation of the local and general condition, morphine was usually given and the patient warmed up. Blood transfusions would have been more frequently used, but we had no typed donors available and so had to be content with intravenous salines. A good many cases did not require any operative treatment, and some cases of through-and-through bullet wounds made a rapid recovery after simple antiseptic dressing, administration of morphine, and rest.

OPERATIVE TREATMENT

In general, it may be stated that we did not find it necessary to carry out the very extensive operative measures advocated by many surgeons during the Great War, and we felt, in fact, that in the few cases in which we performed these extensive and severe operations the mortality was higher than had we done something simpler. There are two factors which have a bearing upon this.

First, the Chinese army fighting here did not possess a highly trained ambulance service as it is understood in western countries, and so we did not see at the base hospitals such a high proportion of badly wounded men as was seen by surgeons in the Great War.

Second, although every wound was an infected one, the virulence of the organisms appears to have been less than that of those met with in France and Flanders. One reads of the high proportion of cases of gas gangrene and tetanus met with during the Great War; here such cases were comparatively rare.

Differences in the bacterial content of the soil doubtlessly account for this. The Chinese make use of human excreta for fertilizing purposes, and, although this practice fosters the spread of intestinal diseases, soil treated by human excreta is freer from the germ of tetanus and the organisms causing gas gangrene than is soil treated with horse excreta.

At the end of the Great War surgeons were advocating very extensive operations for chest wounds. The chest was widely opened under positive pressure anesthesia, and the pleural cavity was cleared of blood and foreign bodies. Wounds in the lung were sought for, excised, and sutured, and even vessels in the lung root were ligatured. Such operations are only likely to produce good results if carried out by a highly trained team. The practice employed here as regards immediate operation was (after the patient's recovery from the initial shock) to excise the wound, removing portions of fractured ribs, to arrest hemorrhage in the parietal wound, to remove foreign bodies if easily accessible, and, if possible, to close the wound by suture. No extensive search for foreign bodies was carried out, and no attempt was made to arrest hemorrhage from the lung itself. The first principle was to close the wound or wounds in the chest wall and to treat the hemothorax later along the lines described below.

With operations for severe thoracoabdominal wounds involving the diaphragm, I have no experience. In these cases it appears that a more extensive operation is essential.

ANESTHESIA

The great majority of our chest operations were carried out under local anesthesia, sometimes supplemented with ether or evipan. For several operations full ether anesthesia was employed. We did not possess any apparatus for giving positive pressure anesthesia.

PNEUMOTHORAX

Open pneumothorax is a familiar finding in chest wounds and is of serious import. There appears, other things being equal, to be a direct relation between the size of the opening and the severity of the symptoms. Clinically the patient is found to suffer from the general symptoms of collapse, with cyanosis and respiratory distress and feeble rapid pulse, and air may be heard entering and being expelled from the wound with the respiratory movements. The treatment adopted was as outlined earlier, i.e., saline infusion for the shock and early production of a closed pneumothorax by suture or packing. I have seen several striking cases where the adoption of these simple measures resulted not only in an immediate improvement of the patient's condition, but in a rapid and complete disappearance of the abnormal conditions inside the chest. An example of this will be given later.

Tension pneumothorax is an extremely interesting and striking phenomenon. The syndrome has been described by John Hunter, who observed it following a sword wound of the lung. Tension pneumothorax may be produced in one of two ways, either by a valvular wound in the chest wall which allows air to enter during inspiration but does not allow it to escape during expiration, or by any condition which allows air to escape into a closed pleural cavity from the lung. The latter sometimes happens when an open pneumothorax is closed or closes itself and the wound in the lung remains open. These conditions result in the development of a positive pressure, first in the pleural cavity of the affected side, and later, if the condition is not relieved, in the pleural cavity of the opposite side. The heart and great veins are subjected to pressure and abnormal lateral movements; the lung on the affected side is wholly collapsed, and on the opposite side, partially compressed. It is easy to understand that very severe symptoms are produced by this state of affairs and that, if unrelieved, it leads to death. I have met with the condition on several occasions —twice following intrathoracic operations, once following a thoracoscopy with adhesion division where presumably the lung had been injured, and twice following chest wounds. The treatment is simple and consists in letting out the air. This can often be done by repeated insertion of a needle, and if this is not enough, a catheter may be inserted into the chest and connected with a bottle of fluid. Frequent readings of the pressure (with an artificial pneumothorax apparatus) are instructive and helpful.

HEMOTHORAX

Hemothorax very commonly accompanies chest wounds and may be serious because of the severity of the initial bleeding or because the blood becomes infected at a later date. The source of the blood may

be from the chest wall or from the lung. Wounds of the heart or great vessels are likely to produce fatal hemorrhage and need not be considered here. The main chest wall vessels which may bleed are the intercostals and the internal mammary.

Hemorrhage is responsible for a large proportion of deaths, which take place soon after the injury. In the initial operative treatment of chest wounds careful search was made for bleeding vessels in the chest wall, and these were picked up and tied. Having dealt with the bleeding from the chest wall, the wound was then closed and no attempt made to search for or to suture a bleeding wound in the lung. It was felt that if the wound involved a large vessel at the root of the lung, death was almost certain anyway, but that if the wound involved only peripheral vessels, these would be controlled by the pressure of the hemothorax and pneumothorax. This was found to be the case. As stated before, the policy adopted was to convert an open pneumothorax or hemopneumothorax, as it nearly always was, into a closed one as soon as possible and to treat the hemorrhage and infection by simpler and safer measures at a later date. Aspiration was not done for several days unless signs of pressure or infection appeared early and forced one to act at once. The reason for the delay was to allow the lung to remain collapsed for a period long enough for the wounded vessel to heal. Too early reduction of pressure might allow the re-expanded lung to start bleeding again. So after waiting a few days for the patient's condition to improve, blood was aspirated, never more than 1,000 c.c. at a time, and air replacement was done. In several cases this treatment resulted in a complete disappearance of the hemothorax without the appearance of any signs of infection. In the majority of cases, however, signs of infection appeared; the patient would begin to have fever, and the blood, on being aspirated, would give evidence of a change to pus either by its appearance or its odor. In no case was a thoracotomy done for hemothorax alone, and this treatment would seem to be unnecessary and harmful. An interesting suggestion has been made by Allen, that hemothorax cases be treated by nursing the patient with the wound dependent so that the blood leaks out through the wound and the hemothorax disappears. He argues that the gradual removal of the fluid by a process of leaking out is less disturbing to the lung than when fluid is aspirated, and that there is, therefore, less likelihood of a recurrence of the hemorrhage. He appears to have had good results in the published series of cases, but it will be noted that in most of them the wounds were small, revolver or stab wounds, and as has been remarked earlier in this paper, it is often remarkable how little damage may be done by the passage of a small projectile through the chest cavity. It is prob-

able that Allen's cases would have done well with any simple treatment. This method of treatment was originally suggested by John Hunter.

PYOTHORAX

This was a very common complication and usually supervened on a hemopneumothorax. In treating these cases the principles laid down by Graham were followed as far as possible. Graham's principles are well known and are as follows:

1. Drainage, but with avoidance of an open pneumothorax during the formative stage of empyema.
2. Early sterilization and obliteration of the cavity.
3. Maintenance of nutrition of the patient.

Graham's methods have the great advantage of simplicity, and no complicated apparatus is required. There is no need to go into the details of treatment of pyothorax in these patients. It is enough to say that the methods used were found very satisfactory and that, out of thirty-five patients with empyemas, all but three were healed at the end of four months. In four patients phrenic evulsions were performed and resulted in an immediate diminution of the size of the cavity (usually to the extent of from 100 to 150 c.c.).

In one patient, after the employment of all simpler measures, the cavity still refused to close and some form of thoracoplasty was being considered, but the man's general condition steadily deteriorated and he died.

Lung abscess sometimes complicated chest wounds. The abscess can usually be drained through the original wound.

Retained Projectiles in the Chest.—A bullet or shell fragment in the lung does not, as a rule, give rise to troublesome symptoms, but if it does it can usually be readily removed. Most of the soldier patients with whom we had to deal have disappeared as far as a follow-up is concerned, so it is impossible to comment upon their afterhistories.

Mortality.—These figures taken from the No. 2 Base Hospital, Shanghai, are fairly representative of the incidence and mortality of chest wounds here.

Total admissions	1,186
Chest wounds	115
Deaths from chest wounds	17

Chest wounds, therefore, formed approximately 10 per cent of the total wounded, and the mortality from chest wounds was 14.8 per cent.

This is a low mortality for wounds caused by modern projectiles, and the explanation probably lies in the fact that a large number of

those with chest wounds died in the field or during transport, and that the base hospitals never saw a high proportion of the very serious wounds.

Traumatic Aneurysm of the Axillary Artery.—A Chinese farmer was admitted three months after being shot, with an entrance wound below the angle of the right scapula and no exit wound. He had a swelling larger than a closed fist below the outer end of the right clavicle and extending into the axilla. The swelling fluctuated but had no pulsation, nor could a bruit be heard. The radial pulse was normal and the circulation in the arm excellent. X-ray examination showed the bullet lodged below the junction of the outer and middle thirds of the clavicle.

His history stated that, following the injury, he had hemoptysis and that the swelling in front appeared very rapidly. He also noticed that for some days the right hand was blue and cold.

Operation.—The second part of the subclavian artery was exposed by an incision above the clavicle, by the method to be described, and a tape passed round it. Next, the third part of the axillary artery was exposed and a tape passed round it. These tapes were given to an assistant, and the swelling itself was exposed and cut into. It was filled with blood clot and was lined with smooth shining membrane. It was being fed by a vessel from above, and this opening was closed by a fine catgut suture on an intestinal needle. The cavity was in part obliterated by the method of Matas, but in some places the walls could not be drawn together and so the remainder was packed with gauze. When the gauze was removed a few days later no hemorrhage occurred. The bullet was inaccessible and was not removed. The man made a quick recovery.

Note on Exposure and Ligature of the Subclavian Artery.—I have on several occasions been impressed by the lack of room between the brachial plexus and the clavicle and the difficulties of access to the third part of the subclavian artery in the classical approach described in operative surgery books. This is commented upon by Cokkinis.* To improve access he recommends division of the clavicle. On the other hand, while doing scaleniotomies, I have often been struck by the ease of access to the second part of the subclavian after division of the scalenus anticus. An incision is made above the clavicle, and the scalenus anticus is exposed and cleaned. The phrenic nerve is mobilized and drawn medially out of harm's way. A Moynihan cholecystectomy forceps is passed under the scalenus anticus. This instrument has just the right shape for this procedure, and its blunt points make damage to the underlying vessel very unlikely. The blades of the forceps are widely opened, and the muscle is raised on them and divided with successive snips of the scissors. The upper end of the divided muscle at once retracts, and the second part of the subclavian artery becomes readily accessible.

SUMMARY

A brief account of the history of this subject is given.

The methods of treatment used in dealing with 150 of these cases are described.

*Post Graduate Surgery, Vol. 3, p. 4090.

Very radical operations were not found necessary, and explanations for this and for the comparatively low mortality are given.

A method of exposing the subclavian artery in the neck is described. I have not seen this method described or practiced elsewhere and believe it to be original.

EDITOR'S NOTE.—It will be interesting to see whether or not the present war will change our ideas of the treatment of gunshot wounds of the chest. It seems hardly likely that the lessons which were so dearly learned in the last war will undergo any very radical changes. Yet new kinds of missiles or new methods of treatment may be introduced. At any rate, the experience of Dr. Ranson in China is interesting as one of the first, if not the first, accounts of gunshot wounds of the chest in the present conflict, the second phase of the great World War.

REFERENCES

1. Hunter, J.: Works, London, 1835.
2. Otis: In Medical and Surgical History of the War of the Rebellion (1861-1865), Washington, 1875.
3. Elliott and Gask.: In Official History of the Great War, Medical Services, Surgery of the War, London, 1922.
4. Lejars: Urgent Surgery, Bristol, 1923.
5. Sauerbruch and O'Shaughnessy: Thoracic Surgery, London, 1937, Arnold.
6. Graham, Singer, and Ballon: Surgical Diseases of the Chest, London, 1935, Lea and Febiger.
7. Sellors, Holmes: Surgery of the Thorax, London, 1933, Constable & Co.
8. Keen's Surgery: Vol. 4, Sections on Military and Naval Surgery, Philadelphia, 1916.
9. Nelson's Loose Leaf Living Surgery.
10. Allen, D. S.: The Treatment for Penetrating Wounds of the Pleural Cavity, Arch. Surg. **21**: 1161, 1930.
11. Graham, E. A., and Berck, M.: Principles Versus Details in the Treatment of Acute Empyema, **98**: 520, 1933.
12. Lockwood, A. H., and Nixon, J. A.: War Surgery of the Chest, Brit. M. J. **1**: 1918.

SELF-RETAINING SCAPULA RETRACTOR

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ELEVATION or displacement of the scapula is frequently required in providing adequate exposure in many thoracic operations. The usual method of accomplishing this is by manual effort, with or without the use of a large-bladed retractor. The task is no easy one for the assistant, should the operation upon the chest wall or within the thorax be time-consuming. This is especially true when upper lobe mobilization is combined with costatectomy in the performance of upper selective thoracoplasty. Bethune[1] designed an "iron interne," which consists of an upright post attached to the operating table, a crossbar or forearm, and a metal hand.

During the past four years, we have used a similar mechanical retractor which embodies the principal features of the Bethune retractor but has the advantage of being more quickly assembled, adjusted, and dismantled. The instrument was constructed by Mr. Howard Pringle,* and valuable suggestions were made by Dr. Albert D. Haug. Several improvements were made upon the original before the present instrument was developed. This mechanical retractor has aided us so materially and queries concerning it by visiting surgeons have been so frequent that a description of the instrument is given for those who may be interested.

The blade (*B*) of the retractor is attached by a swivel to a chain, which is, in turn, wound upon a reel (*R*). The chain is shortened by winding the reel as one winds in a fishing reel. A trigger (*T*) release is provided for lengthening the chain. A post on the bottom of the reel slips readily into the end of a hollow rod which is of adjustable height and is fastened to the operating table. All of the parts shown in Fig. 1, except the clamp for the rail of the operating table, are sterilized by boiling. At the appropriate time in the course of the operation, the assistant slips the rod down through a hole in the sterile drapes, and the rod is fastened in place readily by a nurse or the anesthetist. The reel is then slipped into place, the blade caught about the scapula, and the reel tightened. A major objection to any form of mechanical retractor is the inflexibility of traction applied to the blade. A shift in the position of the patient which alters the relation of the scapula to the fixed upright supporting rod would vary the amount of traction

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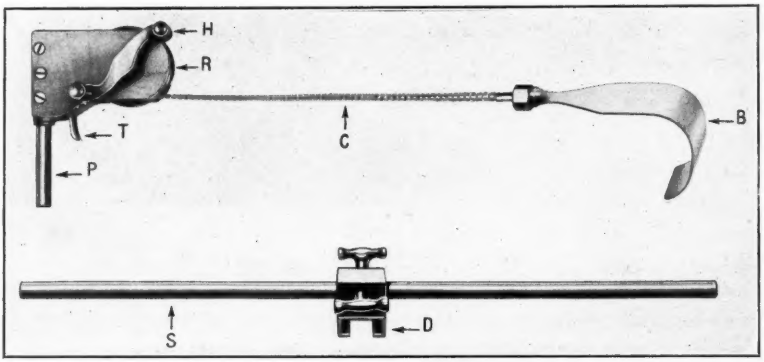

Fig. 1.—Photograph of self-retaining scapula retractor with adjustable reel and chain for the control of traction. *B*, blade; *C*, chain; *R*, reel; *H*, handle for winding reel; *T*, trigger; *P*, reel post; *S*, supporting rod; *D*, clamp for rail of operating table. All parts except the clamp are sterilized by boiling.

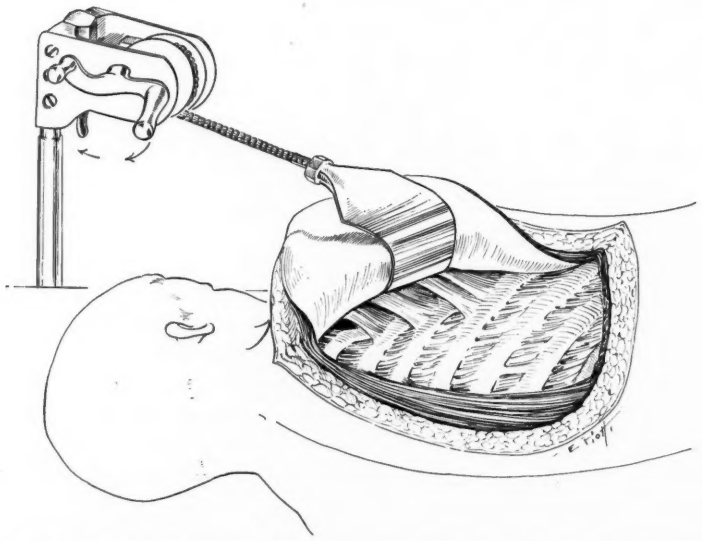

Fig. 2.—Drawing illustrating self-retaining retractor in use. The instrument is readily adjustable. The direction of pull is determined by the position of the clamp on the rail of the operating table and by the elevation of the supporting rod. The degree of traction is controlled by the winding arm of the reel. Adjustments may be made quickly and easily. The instrument may be rapidly assembled and dismantled.

on the scapula. The use of a reel and chain largely does away with this objection. Adjustments may be made easily and quickly by the assistant, either by giving the reel a turn or by using the trigger release as occasion demands.

The entire apparatus may be rapidly dismantled and removed so that it will not be in the way of the assistant during the period of wound closure. The trigger releases the chain, the reel is lifted out of the upright tube, and the tube is dropped out of the field by a simple turn of the screw on the clamp.

REFERENCE

1. Bethune, N.: Self-Retaining Scapula Lifter "The Iron Interne," Instrument Catalogue of George P. Pilling & Son, Philadelphia, Pa., 1932-3, p. 247.

1101 Beacon Street
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PUTRID LUNG ABSCESS

Pathogenesis, Prognosis, and Treatment

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THERE is a great deal of discussion at the present time concerning the treatment of abscess of the lung. It is held by many that the problem is essentially a medical one and requires only conservative measures. This group believes that resolution occurs in the majority of instances and that surgery is the procedure of last resort. The opposing group maintains that medical treatment is a snare and a delusion; that the improvement with conservative measures is transitory and part of the evolution of the disease; and that spontaneous cure is rare and recurrent exacerbations, frequent. The second group further states that the apparently irreconcilable viewpoints are due to two main factors. The first is the failure to differentiate clearly between putrid lung abscess, i.e., abscess with foul secretion, and the various other types of pulmonary suppuration. The second is brief observation and incomplete follow-up of the medically treated cases.

In an attempt to add further information to this controversial subject a study was made of all the cases of putrid lung abscess that were discharged from the medical, surgical, and pediatric wards of the City Hospital between the years 1920 and 1938, inclusive. The other varieties of lung abscess, such as those occurring in bronchiectasis, aputrid necrosis complicating pneumococcus pneumonia, Friedländer pneumonia, or miliary abscesses following bacteriemia, were not included in this study. Tuberculosis was ruled out in all instances by sputum examination, guinea pig inoculation, or necropsy.

There were ninety-four patients with putrid lung abscess treated in the nineteen years included in the survey. Seventy were male patients and twenty-four were females, a ratio of approximately 3 to 1. The age incidence ranged from 2½ to 77 years, but two-thirds of the cases occurred in the fourth, fifth, and sixth decades of life and one-third in the fifth decade alone (Chart 1).

ETIOLOGY

Despite the excellent clinical and experimental studies in the literature, the etiology of putrid lung abscess still remains obscure. There is, however, general agreement that putrid abscess is often preceded by a surgical procedure. On this basis we have arbitrarily divided the cases into two main groups, postoperative (I) and nonpostoperative (II).

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There were fifteen cases in Group I. Ten, or 67 per cent, followed surgery of the tonsils, teeth, jaw, or nose. The five remaining cases followed appendectomy, herniotomy, or mastoidectomy. The type of anesthesia used was not stated in sufficient instances to be informative. There were seventy-nine cases in Group II. Among these were isolated instances which followed aspiration of a staple and of ammonia, submersion, hemiplegia, and acute ulcerative tonsillitis. But in the vast majority of cases in this group, 94 per cent, the abscess was not preceded by any known disease, accident, or operation (Table I).

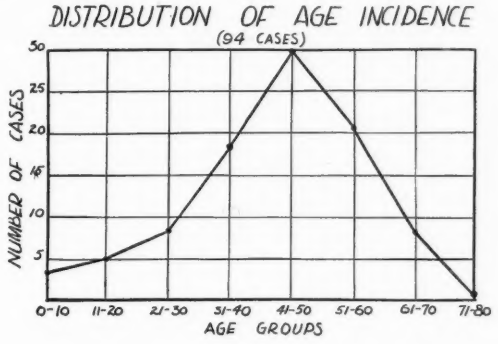

Chart 1.

The relation of operations to putrid lung abscess in our series is low. In only 16 per cent of our cases was there a history of a previous operation. Other reports in the literature show a much higher incidence. For example, Cutler and Gross[1] found a 54 per cent incidence, and Flick,[2] a 70 per cent incidence. Our low rate may be due to a smaller number of tonsillectomies inasmuch as this procedure is quoted as the most frequent cause of abscess. The most significant finding, however, is that 84 per cent of the cases had no previous surgical procedure and that 79 per cent had no predisposing condition whatsoever.

In a report of forty-five cases by Neuhof and Touroff,[3] 31 per cent developed after surgery, and 58 per cent were of uncertain etiology. These authors were of the opinion that the gross infection of the teeth and gums which the patients showed was the predisposing factor in the production of the pulmonary abscess. This was further confirmed by Stern[4] who found that 84 per cent of the cases in which the etiology of the abscess was uncertain had marked pyorrhea and dental infection. Flakes of tartar removed from these teeth showed organisms on smear and culture very similar to those found in lung abscesses. In the City Hospital series there are detailed reports of the condition of the teeth and gums in sixty-two patients. Eighty per cent were markedly carious and had associated gingival infection. This percentage was identical for the cases which followed surgery as well as for those of un-

TABLE I

ETIOLOGY OF PUTRID LUNG ABSCESS

SURGICAL PROCEDURE	NUMBER OF CASES
Group I (Postoperative)	
Tonsillectomy	6
Dental extraction	2
Drainage for osteomyelitis of mandible	1
Nasal operation	1
Appendectomy	3
Mastoidectomy	1
Herniotomy	1
	15 (16%)
Group II (Nonpostoperative)	
No previous condition	74
Acute ulcerative tonsillitis	1
Hemiplegia	1
Submersion	1
Aspiration of ammonia	1
Aspiration of staple	1
	79 (84%)

known etiology. The aspiration of septic tartar into the lungs as a cause for the development of putrid lung abscess deserves serious consideration.

PATHOGENESIS AND PATHOLOGY

In a large number of instances upper respiratory infection, pneumonia, and pleurisy were recorded as precursors of the abscesses. Careful analysis, however, leads us to conclude that these were not separate entities complicated by lung abscess, but were rather early manifestations of the abscess itself. A brief review of the pathogenesis and pathology of putrid lung abscess will make evident the difficulties of early diagnosis.

According to Neuhof and Wessler,[5] the disease is bronchiogenic in origin and begins in the smaller bronchi at the site of the aspirated infectious material. Because of the presence of pathogenic anaerobic organisms, an intense necrotizing process is initiated which involves the surrounding pulmonary parenchyma and produces an inflammation of the overlying pleura. The clinical picture at this stage is one of pulmonary consolidation or pleurisy, and even the roentgenologic film is indistinguishable from that of an ordinary pneumonia. Because of the necrotizing character of the process there is rapid destruction of the bronchi, blood vessels, and pulmonary parenchyma, and a localized gangrenous abscess results. Within one to two weeks after the onset the cavity begins to drain by way of the patent bronchi, and the patient expectorates foul pus. At this time the diagnosis is apparent clinically, and the roentgenologic film usually shows a fairly large cavity.

There are many authors who disagree with the aspiration theory and feel that the disease is embolic in origin. However, analysis of many series of cases in the literature has presented very meager evidence in support of this view. In this series there was not a single case which definitely suggested a hematogenous source of infection. On the other hand, the frequent association of tonsillectomy, dental extraction, and gingivodental infection gives strong support to the aspiration theory. While there is no absolute proof that the disease is developed by anaerobic bacteria, they are practically always present and are the only organisms found in putrid lung abscess which produce gangrenous pulmonary lesions experimentally by bronchiogenic infection. Anaerobic cultures of pus obtained by bronchoscopy have shown many types of organisms to be present. Bucher[6] found anaerobic streptococci and staphylococci, fusiform bacilli, and spirilla. Cohen[7] cultured numerous varieties of anaerobes, but the ones which appeared in greatest frequency were facultative anaerobic streptococci, diphtheroids, and *Bacillus melaninogenicum*. Weiss and Mercado[8] found facultative anaerobic streptococci, *B. melaninogenicum*, spirochetes, and fusiform bacilli. In addition to the anaerobic bacteria, many types of aerobic organisms have also been found. In this series of cases very few special bacteriologic studies were made. Routine smears and cultures of sputum or aspirated pus in about one-third of the cases showed streptococci, both aerobic and facultative anaerobic, fusiform bacilli, spirochetes, and unidentified gram-negative bacilli.

CLINICAL FEATURES

There were no obvious differences noted in the mode of onset between the cases with previous operation and those with unknown etiology. The earliest symptoms were usually those of an upper respiratory infection and they consisted chiefly of productive cough with mucoid expectoration and slight fever. It is of interest that many patients in the unknown etiology group gave a definite history of exposure to inclement weather the day before the onset of these symptoms. In a smaller group of patients the disease began abruptly with chills and fever, followed by productive cough shortly afterward. In another small group the outstanding initial symptom was sudden chest pain. In one case the disease began with a large pulmonary hemorrhage. After a week or two of illness, the symptoms in all cases were very similar and differed only in degree of severity.

The one symptom common to all these cases was cough with the expectoration of foul sputum. The foul expectoration usually appeared within two weeks of the onset of illness and occurred suddenly after a severe coughing paroxysm. In the majority of patients the sputum was also blood-streaked, and not infrequently there were moderately severe pulmonary hemorrhages. Chest pain was another char-

acteristic symptom. It was generally severe and affected by respiratory movements. In practically all cases the location of the pain coincided with the side of the chest involved, and very often it was most severe over the site of the abscess itself. The pleuritic origin of the pain is the obvious explanation for this phenomenon. The temperature was markedly elevated during the first weeks of illness, but after the expectoration of foul pus it usually subsided and continued subfebrile. Toxicity was present in varying degrees and was most marked in the patients with pleural perforation. Dyspnea and weakness were fairly constant features. In patients with prolonged illness there was marked loss of weight.

The physical signs of the disease varied with the stage of the process. At the onset the signs were typical of a lobar pneumonia. As liquefaction necrosis developed, the signs became more suggestive of cavitation. In general, however, dullness, bronchovesicular breathing, and coarse râles were the most constant findings. Cavernous breathing and pectoriloquy were not usually present until the later weeks of illness. The precise location of the abscess by physical signs was usually rendered difficult because of the associated presence of sibilant râles in apparently uninvolved parts of the lung. In a limited number of instances there were tender areas on the chest wall overlying the site of the abscess. Bronchoscopy was of the greatest assistance in diagnosing the exact source of the suppuration, either by aspiration of foul pus from the involved lobar segment, or by disclosure of an edematous bronchus which led to the diseased part.

Hematologic examinations generally showed leucocytosis and increase in polymorphonuclear cells. At the onset of illness the leucocyte counts often ranged between thirty and forty thousand cells per cubic millimeter and gradually decreased as the disease entered upon the subacute and chronic phases. Many patients showed marked secondary anemia, and hemoglobin determinations below 50 per cent were not at all uncommon. Blood chemistry examinations showed no changes which could be ascribed to the disease in particular. Small amounts of albumin were found in the urine in many of the patients.

The roentgenologic examination was obviously the most important diagnostic procedure. Although the films at the onset of illness usually suggested pneumonic consolidation, not infrequently there were noted small areas of rarefaction. As the disease progressed the zone of consolidation receded, and evidence of cavitation with fluid level became more noticeable. In the subacute and chronic phases the typical picture was that of an isolated large abscess limited to a pulmonary segment. Bronchography with lipiodol was often used in these cases to demonstrate the size of the cavity and the extent of the associated bronchiectasis. Occasionally the routine film would show apparent recovery, whereas lipiodol studies would demonstrate residual cavitation.

The distribution of the abscesses in this series according to lobes is shown in Table II.

TABLE II

LOBAR DISTRIBUTION OF ABSCESSES

LOBE	NUMBER OF CASES	
Right upper	29	
Right middle	2	61
Right lower	30	
Left upper	13	35
Left lower	22	

In all cases but two the abscess was localized to a single lobe. In the two multiple cases the abscesses were in the right upper and middle lobes, and in the right upper and left upper lobes, respectively. Since exact localization of the abscesses was not attempted until the later years covered by this study, it is not possible to give an accurate distribution of the lesions according to bronchopulmonary segments. In general, however, most of the lesions in the lower lobes were situated in the apical segments. The incidence of right-sided lesions was predominantly greater than that of left-sided ones, but there was almost equal distribution of lesions between the right upper and right lower lobes. On the left side the lower lobe abscesses predominated. Review of case reports by Flick,[2] Neuhof and Touroff,[3] and Cutler and Gross[1] also shows preponderance of right-sided lesions. This has been suggested as an argument for the aspiration theory inasmuch as the right main bronchus is the more direct communication from the trachea. The high incidence of single abscesses in all series of cases, particularly this one, also supports the aspiration theory. There is, however, one objection which is suggested by these same statistics. If the aspiration is the mechanism of infection, then the lower lobes should be involved to a far greater degree. Nevertheless, in all studies, the distribution between upper and lower lobes is almost equal.

COMPLICATIONS

There were many associated diseases among the ninety-four cases of putrid lung abscess. About 10 per cent of the patients had syphilis, and approximately an equal number had hypertensive or arteriosclerotic cardiovascular disease. Three patients were diabetics. The other complicating conditions were varied and occurred only in individual instances.

The most important complication of the abscess itself was perforation into the pleural cavity with production of a putrid empyema. This occurred in 12 per cent of the cases. Most of the empyemas were small and encapsulated. In two cases there were large perforations with marked pyopneumothoraces. Cerebral metastases occurred in 3

per cent of the cases. Serous effusions were present in two instances, one after operation. Terminal bronchopneumonia occurred three times. Despite the chronicity of the disease in many of the cases, amyloidosis was found only once, on necropsy examination. While there were numerous instances of extension of the abscess within its original pulmonary lobe, there were only three instances in which the abscess spread from one lobe to the adjoining one across the interlobar fissure. There were two instances of contralateral metastasis. Bronchopleural fistulas were present in all the operated cases.

MEDICAL TREATMENT

Of the ninety-four cases in the series, seventy-two were treated medically and twenty-two were treated surgically. The medical procedures included postural drainage, bronchoscopic aspiration and irrigation, artificial pneumothorax, intravenous injection of arsphenamine and of guaiacol, rectal injection of ether in oil, and oral administration of potassium iodide and sulfanilamide. One of the cases had been previously treated unsuccessfully with lobectomy and thoracoplasty at another institution. The duration of illness of the medical cases ranged from 16 to 3,217 days, but the majority of cases were sick less than 150 days. The number of days of hospitalization ranged from 1 to 274 days, with the majority of cases hospitalized less than 40 days. Approximately one-fourth of the patients had been previously at City Hospital or at other institutions for the treatment of lung abscess. Nine patients were hospitalized twice; four were hospitalized three times; and four were hospitalized four times. The condition on discharge of the seventy-two medically treated cases was as follows: Cured, four cases (5 per cent); improved, twenty-two cases (31 per cent); unimproved, twelve cases (17 per cent); dead, 34 cases (47 per cent). More than one-third of the patients who died were in the hospital less than ten days, and two-thirds were in the hospital less than twenty days.

In summarizing the condition on discharge of the seventy-two patients we accepted the final status as described by the house staff physician. However, if we analyze these results according to definite criteria, we find it necessary to make many changes. These criteria are as follows:

1. Cured: No symptoms and no roentgenologic evidence of abscess.
2. Improved: No symptoms and roentgenologic evidence of a retrogressing lesion.
3. Unimproved: Symptoms present, or no symptoms but roentgenologic film showing abscess to be stationary or progressive.

Of the twenty-two cases discharged as improved, only three qualified according to these standards. Study of the other nineteen cases showed

that seventeen were discharged with roentgenologic evidence of either a stationary or a progressive lesion and that two were discharged with characteristic symptoms of abscess. If we then revise the statistics according to the criteria described above, we find the end results of medical treatment in the seventy-two cases as shown in Table III.

TABLE III

RESULTS OF MEDICAL TREATMENT

STATUS	NUMBER OF CASES	
Cured	4 (5%)	9%
Improved	3 (4%)	
Unimproved	31 (44%)	91%
Dead	34 (47%)	

Study of the case histories indicates that most of the patients discharged as unimproved develop frequent exacerbations of the abscess and eventually succumb to the disease. In a few instances the necrotizing process finally subsides, but the patient is left with extensive bronchiectatic disease at the previous site of the abscess and also in other parts of the lung. This point is well demonstrated in the following case report:

G. B. (No. 7648), a 58-year-old negro, was admitted on Dec. 4, 1928, for the treatment of extensive burns of the back. He had been treated for lung abscess on the right side nine years previously but since then had been relatively well except for bloodstreaked expectoration. The patient died of a left upper lobe pneumonia and acute nephrosis six weeks after admission. Autopsy examination disclosed marked dilatation of all the bronchi of the right lung with large bronchiectatic cavities containing nonfoul pus. Between the bronchi there was a chronic interstitial pneumonitis. (This case was not included in the series because of the complicating factors).

There were five patients in the unimproved group in whom the clinical duration of illness at the time of discharge exceeded five years. It is not unlikely that in these patients the anaerobic infection will eventually subside, leaving only extensive bronchiectatic changes as were observed in the recently cited case of G. B. In over 60 per cent of the unimproved patients the duration of illness did not exceed one year. What the prognosis is in this group can best be surmised by studying the duration of illness in the patients who died. There were thirty-four deaths, but in two of the charts the duration of illness preceding hospitalization was not given. Of the remaining thirty-two cases, there were twenty-five (78 per cent) in whom the entire duration of illness, from onset to death, did not exceed one year. In seven patients (22 per cent) the duration of illness was greater than one year, but in only three instances was it greater than two years. The longest duration of illness was nine years and the shortest was nineteen days. The latter case had been in the hospital for treatment of diabetes

for a period of three months prior to the onset of the lung abscess, and the entire progress of the disease was observed.

There is a marked discrepancy between the results at City Hospital and those described in several representative series in the literature. In contrast to the 9 per cent cured or improved, 44 per cent unimproved, and 47 per cent dead, there are the results of Cutler and Gross,[1] who reported a series of thirty-eight cases with 53 per cent cured and 28 per cent dead. Allan and Blackman,[9] in a study of fifty-five cases, reported 54.5 per cent well and 25 per cent dead. Flick and his associates,[2] in a series of 121 cases, reported 54 per cent well and only 2.4 per cent dead. Brunn[10] collected 205 cases from four hospital services and found 31 per cent improved and 35 per cent dead. It is probable, however, that the differences between our results and those reported elsewhere are more apparent than real. Had we been less exacting in our criteria we would have accepted the original number of improved cases, and our end results would have been 36 per cent cured or improved. Our statistics are not the only ones which give a poor prognosis for the conservative treatment of lung abscess, for Lord[11] reported a 75 per cent mortality rate in a series of ninety-seven cases.

Careful study of the records of the patients in this series has led us to conclude that, unless definite criteria for cure are established, many erroneous discharge diagnoses will be made. This is even more important in the patients discharged as improved. The patient with putrid lung abscess will, in the majority of instances, show a marked clinical improvement once bronchial drainage is established. After a few weeks the temperature may become subfebrile or normal, the sputum may become scanty, and the general condition of the patient appear greatly improved. However, these symptomatic improvements cannot be taken as indices for the eventual prognosis. Unless the roentgenologic film shows definitive clearing of the process, the patient may have recurrences of the abscess at any time later. It is only after prolonged follow-up of the cases that one can really determine the final outcome of the disease. The average duration of hospitalization is usually too brief to enable the observer to formulate a definite opinion of the fate of the patient. Several case histories are presented as illustrations.

CASE 1.—F. H., No. 53654, a 25-year-old white man, was admitted to the City Hospital on March 7, 1935. His chief complaint was pain in the left chest of three days' duration and productive cough of one week's duration. Examination on admission revealed dullness and diminished breath sounds over the left lower lobe. The temperature was 100° F. X-ray examination showed fluid and consolidation at the left base (Fig. 1). The clinical diagnosis was pneumonia. The temperature subsided rapidly, and the patient was discharged as improved on March 20, 1935, thirteen days after admission. He was readmitted on March 22. In the two-day

Fig. 1.—Case 1, March 9, 1935. Fluid and consolidation present at left base.

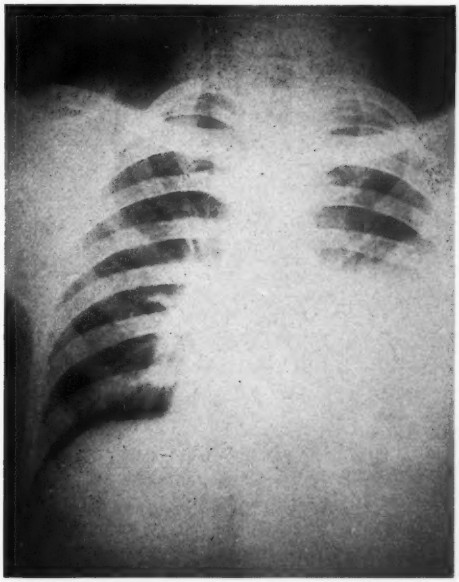

Fig. 2.—Case 1, April 2, 1935. Fluid and consolidation increased.

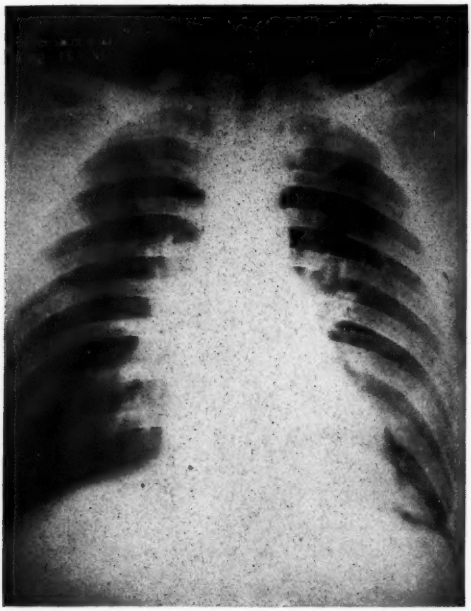

Fig. 3.—Case 1, Dec. 21, 1935. Fluid and consolidation no longer present. X-ray shows only fibrotic infiltrations in the left lower lobe.

Fig. 4.—Case 1, Jan. 10, 1936. Bronchogram with lipiodol demonstrating extensive cylindrical bronchiectasis of the left lower lobe.

interval at home his sputum had become foul and he had again become febrile. Roentgenologic examination disclosed more consolidation and fluid at the left base (Fig. 2). Aspiration of the fluid showed it to be purulent. The patient was treated with chest aspirations and ether-in-oil enemas. His temperature subsided, and he was again discharged as improved on June 24, 1935. He was admitted for the third time on Dec. 20, 1935, with the history that, following his discharge six months previously, he had continued to cough and that the day before admission he had had chills, fever, and hemoptysis. Examination showed fibrotic infiltrations in the left lower lobe (Fig. 3), and lipiodol studies demonstrated extensive cylindrical bronchiectasis (Fig. 4). The patient was treated with bronchoscopic irrigations of saline and 20 per cent argyrol, injections of neosalvarsan, and artificial pneumothorax. The symptoms subsided and he was again discharged as improved on Feb. 13, 1936. His fourth exacerbation occurred in September, 1937, and he died in another hospital on March 9, 1938.

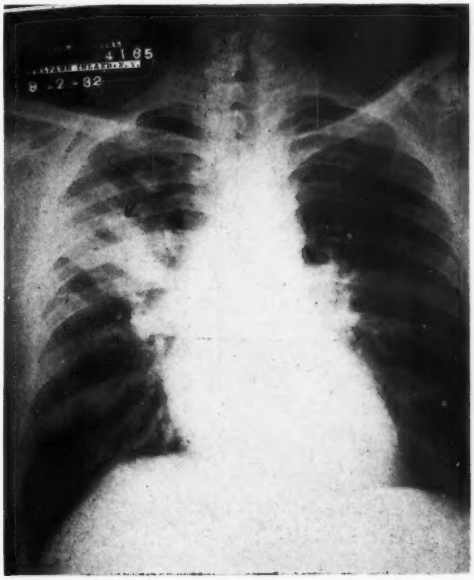

Fig. 5.—Case 2, Sept. 2, 1932. Consolidation and multiple cavitation in the right upper lobe.

Case 2.—T. L., No. 34385, a 24-year-old white man, was admitted on Sept. 14, 1922. His complaint was chills, fever, and productive cough of one week's duration. Examination disclosed dullness, diminished breath sounds, and occasional râles over the right upper lobe. The temperature ranged between 99 and 105° F. X-ray examination showed pneumonic consolidation of the right upper lobe. Eight days after admission the temperature began to decline, and the sputum became foul. X-ray examination at this time showed a large cavity in the right upper lobe. The temperature gradually became normal and the sputum odorless. X-ray examination five weeks after admission showed extensive fibrosis, and the cavity was no longer visible. The patient was discharged as improved on Nov. 1, 1922, forty-eight days after admission.

On Aug. 31, 1932, he was readmitted to City Hospital. Since his discharge ten years before he had had recurrent episodes of cough and fever. The last exacerba-

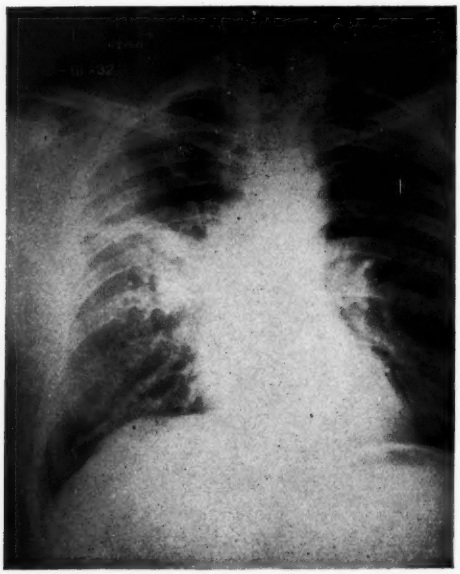

Fig. 6.—Case 2, Oct. 19, 1932. There is no evidence of resolution, and cavitation is more extensive.

Fig. 7.—Case 2, Jan. 19, 1933. There is slight resolution of the inflammatory process, but cavitation is still visible.

tion had occurred five days before admission. Examination disclosed consolidation with multiple cavitation in the right upper lobe (Fig. 5). The temperature was

elevated and the sputum profuse and foul. The clinical picture remained unchanged for about a month, at which time the x-ray examination showed more extensive cavitation (Fig. 6). Shortly afterward, the temperature began to decline, and the patient was discharged as improved on March 30, 1933, after 214 days of hospitalization. His last x-ray, made six weeks before discharge, showed slight resolution of the inflammatory process, but cavitation was still visible (Fig. 7).

CASE 3.—C. M., No. 61113, a 60-year-old white woman, was admitted on Jan. 15, 1936. Her illness had begun eight years previously when, after exposure to cold, she had had a large pulmonary hemorrhage. Since that time, she had had frequent recurrences of productive cough, hemoptysis, and foul expectoration. Examination disclosed dullness and râles at both bases and marked clubbing of the fingers and toes. The temperature was normal. X-ray and lipiodol studies showed saccular and cylindrical bronchiectasis in the right lower lobe. The cough and expectoration diminished, and the patient was discharged as improved on Feb. 11, 1936, twenty-seven days after admission. She was readmitted eight months later on Oct. 10, acutely ill, febrile, and toxic. The expectoration was foul and bloody. X-ray examination showed pneumonic consolidation of the right lower lobe. The clinical course was progressive, and she died on Nov. 22, 1936.

CASE 4.—G. J., No. 10576, a 43-year-old white man, was admitted to Bellevue Hospital on March 31, 1929, with a history of chills, fever, pain in the left chest, and cough of three days' duration. The temperature ranged between 99 and 105° F., and the sputum was foul. Examination disclosed dullness, bronchovesicular breathing, and râles over the left upper chest; x-ray examination showed pneumonic consolidation of the left upper lobe. The clinical course was one of gradual improvement. X-ray examination a month after admission showed cavitation in the left upper lobe, and, two months after admission, fibrotic infiltrations. The patient was discharged as improved on June 14, 1929, seventy-five days after admission. The sputum was clear and odorless, and the temperature was normal. Four days later the patient was admitted to City Hospital acutely ill, and he died on the day of admission.

These cases are fitting examples of the erroneous conclusions one may arrive at by using symptomatic improvement as the index of the condition on discharge in cases of putrid lung abscess. Unless the roentgenologic film shows unequivocal evidence of resolution, the patient should not be considered cured or improved, regardless of the absence of symptoms. As was demonstrated in Case 4, the x-ray film made shortly before discharge showed only fibrotic infiltrations with no apparent cavitation. Yet, two weeks later, the patient developed an exacerbation of the disease from which he died. It is important, therefore, to investigate these cases carefully for residual cavitation. Lipiodol studies are often of great value in this respect. The following case report is illustrative of this point:

CASE 5.—J. B., No. 81161, a 50-year-old white man, was admitted on Sept. 1, 1938, in a moribund condition, with a history of chills, fever, and pain in the left chest for one day. The temperature ranged between 99 and 105° F. The physical signs were typical of a left lower lobe pneumonia, but the x-ray examination suggested cavitation (Fig. 8). Eight days after admission the patient expectorated profuse amounts of foul sputum, and x-ray examination on Sept. 9, 1938, showed giant cavitation in the left lower lobe (Fig. 9). The symptoms gradually subsided, and the pa-

Fig. 8.—Case 5, Sept. 1, 1938. Pneumonic consolidation with areas of rarefaction in the left lower lobe.

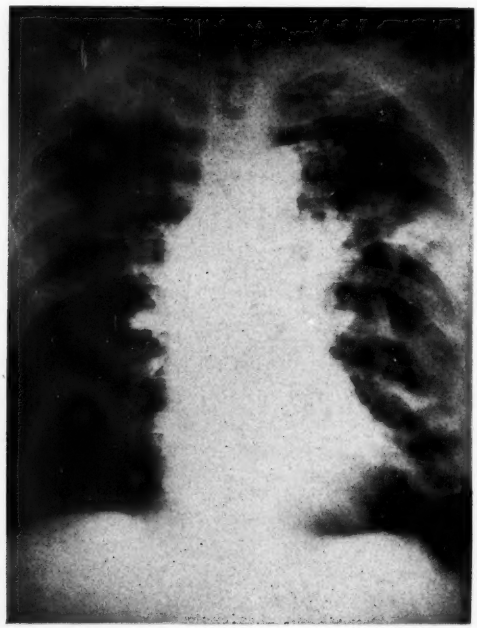

Fig. 9.—Case 5, Sept. 9, 1938. The major portion of the left lower lobe is replaced by several giant cavities.

tient was discharged as improved on Nov. 21, 1938. X-ray examination on October 7 (Fig. 10) showed definite evidence of retrogression and the x-ray on October 19 showed the abscess to be apparently healed (Fig. 11). However, lipiodol study on November 9 showed extensive bronchiectasis and residual cavitation (Fig. 12).

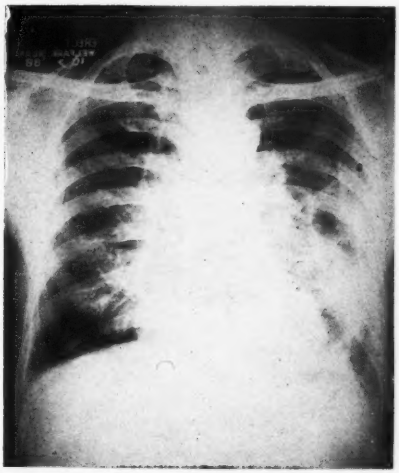

Fig. 10.—Case 5, Oct. 7, 1938. The inflammatory process has retrogressed, and cavitation is less extensive.

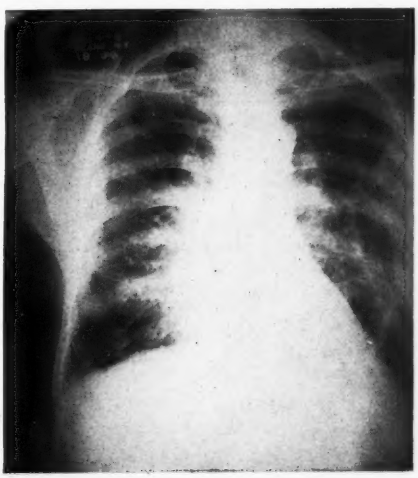

Fig. 11.—Case 5, Oct. 19, 1939. No evidence of cavitation on routine film. Abscess is apparently healed.

Repetition of the lipiodol examination on Jan. 10, 1939, showed the bronchiectasis to be slightly less and the cavity smaller but still present (Fig. 13). At the present time the patient is in excellent condition and has no symptoms of abscess. However,

the fact that the lesion has persisted for four and one-half months makes the final outcome still questionable. It will take many more months of follow-up to ascertain the true result.*

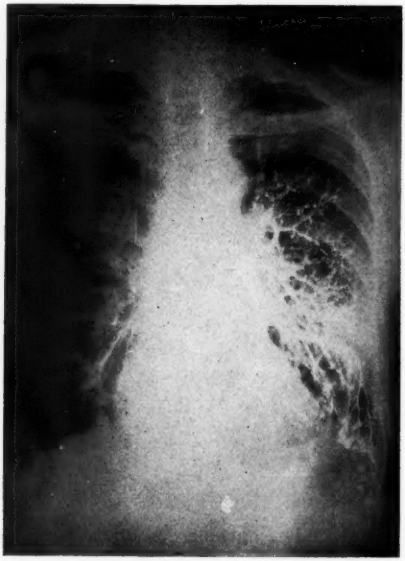

Fig. 12.—Case 5, Nov. 9, 1938. Bronchogram with lipiodol shows extensive bronchiectasis and residual cavitation.

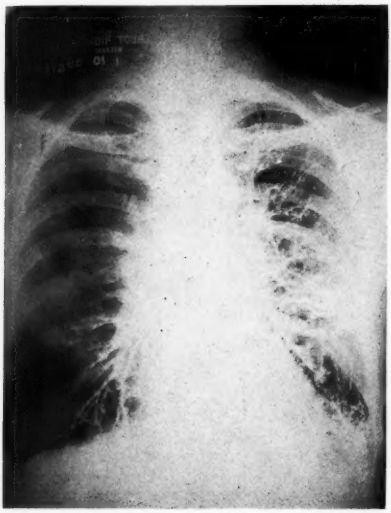

Fig. 13.—Case 5, Jan. 10, 1939. Bronchogram repeated two months later shows bronchiectasis and cavitation to be less extensive but still present.

*Subsequent follow-up in the outpatient department revealed that during the past year the patient has had occasional cough with foul expectoration. Bronchography on Nov. 2, 1939, still showed residual cavitation.

In evaluating the end results of medical treatment in this series we did not accept the discharge diagnosis of improved unless there was roentgenologic confirmation. Review of the cases cited above, as well as of others in the series, illustrates the unreliability of prognosis based on clinical symptoms. The following brief presentations are representative of the cases discharged as improved but which we reclassified as unimproved. The contrast between the status of the disease as determined by the patient's symptoms and as determined by the roentgenologic film is very significant.

CASE 6.—R. B., No. 20645, a 20-year-old white woman, was admitted on Dec. 13, 1930. She presented the typical symptoms and signs of putrid lung abscess. The x-ray examination showed pneumonic consolidation of the right lower lobe with a large cavity in the apical segment. All her symptoms subsided within two weeks, and she was discharged as improved on Dec. 28, 1930. However, an x-ray taken the day before discharge showed the cavity to be unchanged from the film on admission.

CASE 7.—J. W., No. 62922, a 55-year-old negro, was admitted on April 2, 1936, with a history of lung abscess of one and one-half years' duration. The x-ray made upon admission showed consolidation of the right upper lobe (Fig. 14). His symptoms cleared up entirely within a month, and he was discharged as improved on May 6, 1936. The x-ray taken five days before discharge showed multiple cavities in the right upper lobe (Fig. 15).

CASE 8.—J. K., No. 18799, a 67-year-old white man, was admitted on June 30, 1930, with a history of two years' duration. X-ray examination showed a large cavity in the left upper lobe surrounded by an inflammatory zone. After seventy-one days of hospitalization, he was discharged as improved and practically asymptomatic. The x-ray taken two days before discharge showed the cavity to be entirely unchanged.

CASE 9.—J. B., No. 60424, a 42-year-old negro, was admitted on Oct. 21, 1935, with a history of ten days' duration. The admission film showed pneumonic consolidation of the right upper lobe with small areas of rarefaction. Two weeks later the film showed a giant cavity in this region. The patient remained in the hospital for eighty-one days, during which time all symptoms cleared up, and he was discharged on Jan. 10, 1936, as improved. The x-ray film taken a week before discharge showed the cavity to be entirely unchanged, and, in addition, there were new infiltrations in the opposite lung.

CASE 10.—E. S., No. 59437, an 18-year-old white girl, was admitted on June 3, 1935, with a history of two months' duration. The initial x-ray showed pneumonic consolidation of the left lower lobe, and examination one month later showed a large cavity. The patient was in the hospital for 173 days and was treated with many bronchoscopic aspirations. She was discharged as improved on Nov. 23, 1935, with no symptoms. Roentgenologic examination two weeks before discharge showed the cavity to be still present and of the same size as it had been four months previously.

CASE 11.—W. D., a 26-year-old white man, was admitted on Aug. 12, 1927, with a history of four months' duration. Physical signs and roentgenologic examination disclosed a large cavity at the apex of the right lower lobe (Fig. 16). Shortly after admission the temperature subsided, the cough diminished, and the patient was discharged as improved on Sept. 28, 1927, in excellent general condition. X-ray examination two weeks before discharge showed the presence of the original cavity and also of another cavity directly below it (Fig. 17).

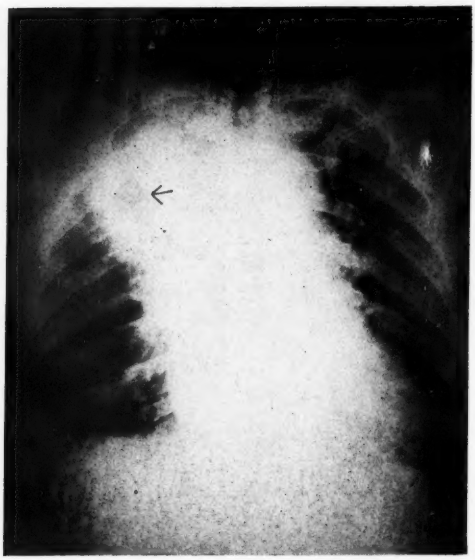

Fig. 14.—Case 7, April 2, 1936. Pneumonic consolidation of the right upper lobe with a large area of rarefaction.

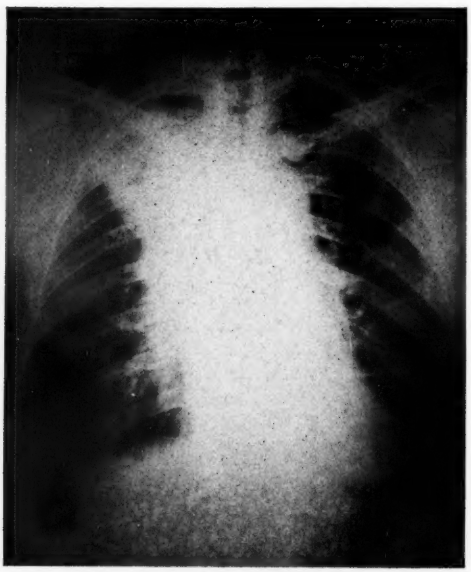

Fig. 15.—Case 7, May 1, 1936. All symptoms entirely cleared, but x-ray shows consolidation and multiple cavitation.

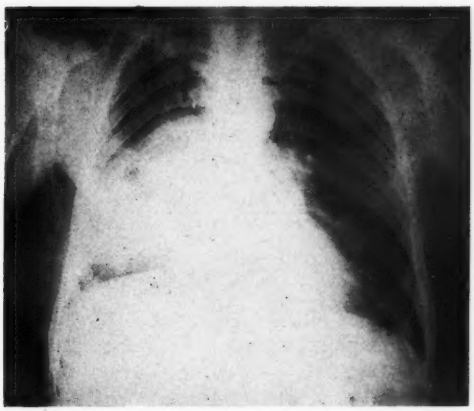

Fig. 16.—Case 11, Aug. 13, 1927. Pneumonic consolidation of the right lower lobe with a large cavity in the apical segment.

Fig. 17.—Case 11, Sept. 10, 1927. No symptoms present, but the x-ray shows cavitation to be more extensive than on admission.

NECROPSY FINDINGS

Analysis of the thirty-four deceased patients revealed the causes of death to be as follows:

1. Progression of the abscess	23 cases	(67 per cent)
2. Cardiac failure	3 cases	(9 per cent)
3. Pulmonary hemorrhage	2 cases	(6 per cent)
4. Cerebral metastases	2 cases	(6 per cent)
5. Bronchopneumonia	2 cases	(6 per cent)
6. Toxic hepatitis	1 case	(3 per cent)
7. Gangrenous esophagitis	1 case	(3 per cent)

Although the majority of cases died either with, or because of, progression of the pulmonary disease, it is important to note that this progression was virtually limited to the lobe originally involved. Only occasionally did the process spread to the adjoining lobes or to the opposite side. This roentgenologic observation was confirmed at necropsy. In view of the relatively small amount of pulmonary tissue usually involved, it would appear that the toxemia produced by the abscess was the primary cause of death rather than respiratory insufficiency.

Necropsy examinations were done on eleven of the thirty-four medical deaths. The findings, in general, were those of a large multilocular abscess with dense pleural adhesions. The adhesions were either localized at the site of the abscess or extended over a large portion of the involved lung. The walls of the cavity were thickened, fibrous, and lined either with a necrotic membrane or with chronic granulation tissue, depending upon the stage of the disease. The cavity, itself, contained foul exudate and debris and, occasionally, blood clots. Leading into the abscess were numerous dilated bronchi containing purulent exudate. In the region of the lung surrounding the abscess there was often a serofibrinous exudate. More commonly the interstitial tissue was fibrotic and friable. Two of the eleven cases showed extension into the adjoining lobes, and one of them also had contralateral miliary abscesses. In the other nine cases the process was limited to the original lobe. The uninvolved lobes showed varying degrees of emphysema, and the contralateral lung was usually negative. The complications found on necropsy were varied. They included isolated instances of bronchopneumonia, empyema, esophagitis, bacterial endocarditis, ulcerative tracheitis, coronary sclerosis, healed apical tuberculosis, adrenal tuberculosis, and amyloidosis.

DIFFERENTIAL DIAGNOSIS

It is not within the scope of this paper to discuss in detail the differential diagnosis of putrid lung abscess. However, we would like to point out that several of the patients in this series had been treated for pulmonary tuberculosis at sanatoriums for periods of as long as three years before the correct diagnosis was established. Of equal significance is the fact that many cases clinically diagnosed as lung abscess were found on necropsy to be cases of pulmonary tuberculosis. These cases obviously were not included in the study. The clinical features of putrid lung abscess are relatively characteristic in the acute and subacute stages, but in the chronic stage the outstanding manifestations are usually those of extensive bronchiectasis rather than of putrid abscess. Neoplasms of the lung with abscess formation are not infrequent diagnostic problems. In Allan's and Blackman's series[9] of 100 cases of lung abscess, five were due to bronchial carcinoma. Most

cases of lung abscess are probably considered as pneumonia at the onset, but the appearance of foul sputum and areas of rarefaction on the roentgenologic film should make the diagnosis apparent. Other conditions to be considered in the differential diagnosis are mycotic infections, Friedländer pneumonia, aputrid necrosis, cystic disease of the lung, and suppurative bronchiectasis.

SURGICAL TREATMENT

Twenty-two of the ninety-four patients were treated surgically. In one instance the operative procedure consisted of bronchoscopic removal of a staple which resulted in a cure. However, this case is not included in the general surgical discussion. The duration of illness of the other twenty-one cases was varied and ranged between three weeks and ten years. About one-third of the cases were operated upon within six weeks of illness, and about one-third after one year. The average duration in the hospital prior to operation was forty-five days. One-third of the cases had been previously hospitalized and discharged as improved with medical treatment. External drainage with evacuation of the cavity was the procedure used in all instances but one. In the latter instance, closed thoracotomy was done. Sixteen of the drainage operations were done in one stage; one was done in two stages; and three were done in three stages.

The criteria for determining if a patient was cured on discharge were the same as those for the medical cases, with the additional proviso that the operative wound had to be healed. The results were as follows: Cured, two cases; improved, four cases; unimproved, three cases; and dead, twelve cases. Three of the improved cases were followed in the outpatient department, and within three months they satisfied the criteria for cure. One improved case did not return for a checkup. The final corrected results are shown in Table IV.

TABLE IV

RESULTS OF SURGICAL TREATMENT (21 CASES)

STATUS	NO. OF CASES
Cured	5 (29%)
Improved	1 (29%)
Unimproved	3 (14%)
Dead	12 (57%)

Four of the deaths occurred within twenty-four hours of operation and were due either to shock or pulmonary edema. Two of the deaths occurred within two weeks of operation and were apparently due to emboli. The remaining causes of death were progression of the disease (3 cases), cerebral metastasis (1 case), bronchopneumonia (1 case), and asphyxia due to a mucus plug in the trachea (1 case). Four of the patients came to necropsy. The characteristics of the disease

and the types of complications were essentially the same as those found in the medical post-mortem cases.

It is difficult to compare the above surgical results with those in the literature because of the variety of the operative procedures used by many of the authors. In addition to external drainage, the operations included lobectomy, pneumonectomy, thoracoplasty, and closed thoracotomy. In Brunn's series[10] the results showed 56 per cent improved and 44 per cent dead. Cutler and Gross[1] found 47 per cent cured or improved and 45 per cent dead. Flick, Clerf, Funk, and Farrell[2] found 31 per cent cured and 34.5 per cent dead. Summing up these three reports, we find that, out of 148 surgically treated patients, 45 per cent recovered and 41 per cent died.

The surgically treated cases in this series showed a much higher percentage of cures than the medically treated ones, but they also showed a very high mortality rate. Out of twenty-one cases operated upon, twelve died. However, these deaths were not generally due to progression of the pulmonary disease. For the greater part, they occurred either at operation, directly after operation, or within two weeks of operation. As was stated previously, four deaths were due to shock or pulmonary edema, two were due to emboli, and one was due to asphyxia. In other words, these fatalities may be considered as either operative or accidental and do not accurately represent the effect of surgery on the course of putrid lung abscess. The fact that two-thirds of the patients were operated upon in the subacute and chronic stages undoubtedly contributed to the high operative mortality.

CONCLUSION

The end results of the conservative treatment of putrid lung abscess compel us to conclude that practically all the various therapeutic measures are valueless. Postural drainage aids somewhat in the evacuation of pus, but it in no way contributes to a definite cure. Pneumothorax is definitely contraindicated because of the danger of tearing the pleural adhesions and producing empyemas. Phrenicectomy was not done in this series, but the authors who used the procedure have since abandoned it. The intravenous injection of arsphenamine and other drugs and the rectal injection of ether have never justified their widespread use. Bronchoscopy may help in establishing drainage but has no effect on the abscess itself. We feel that the essential use for bronchoscopy is diagnostic and not therapeutic (except in the removal of foreign bodies).

Whatever success has been achieved with the conservative treatment of lung abscess has been due to spontaneous improvement rather than to the effects of specific therapy. It was repeatedly pointed out in the case histories that symptomatic improvement occurred after the

first few weeks of illness in most cases. This was independent of therapy and was part of the evolution of the disease. However, this improvement was permanent in only a small percentage of cases. In a series of seventy-two patients treated conservatively, only 9 per cent were cured or improved, while 91 per cent were unimproved or dead.

The best results in the treatment of putrid lung abscess are those reported by Neuhof and Touroff.[3] These authors regard the results of medical treatment so unsatisfactory that they consider practically every case as an acute surgical problem. Their preferred operative technique is that of one-stage external drainage with excision and wide packing of the abscess. Out of a series of forty-five consecutive cases operated upon in the acute stage, i.e., within six weeks of illness, they report forty-three cures and two deaths. While it is undoubtedly true that some of these cases might have recovered spontaneously, one cannot minimize the results obtained, namely, 95 per cent cured.

As a result of the experiences in the series of cases at City Hospital and of the general observations in the literature, we feel that the ideal treatment for putrid lung abscess may be summarized as follows. The patient should be observed for a period of approximately six weeks from the onset of illness. During this time he should be given supportive medical treatment. Serial x-rays should be taken weekly to determine the progress of the lesion, and special roentgenologic studies should be made to localize the abscess accurately. Bronchoscopy should be performed routinely to rule out foreign body or malignancy and to assist in further localization. If, at the end of six weeks, there is no roentgenologic evidence of resolution, the patient should be operated upon. Moreover, if during the period of observation there is evidence of perforation or of progression of the abscess, the case should be operated upon without further delay. The observation period of six weeks was arbitrarily chosen because it usually comprises the acute phase of the disease. In certain instances it may be advisable to watch the case longer. However, surgery should not be needlessly delayed, for there is a definite tendency for the abscess to become rapidly multilocular and thus make complete surgical drainage more difficult. The excellent results obtained by Neuhof in operating on acute lung abscess and the poor results of conservative treatment compel us to conclude that, in most instances, prolonged wishful observation is not in the best interests of the patient.

It is to be emphasized that the object of surgical treatment is aeration as well as drainage. Putrid lung abscess is essentially an anaerobic infection, and thorough ventilation is necessary to destroy the organisms. The abscess must be thoroughly excised, all hidden recesses explored, and the wound packed widely and prevented from closing prematurely.

TABLE V

CASES OF PUTRID LUNG ABSCESS

CASE NUMBER	AGE	SEX	ETIOLOGY	DURATION (DAYS)	LOBE INVOLVED	TREATMENT	RESULT
344	38	M	Unknown	66	R. U.	Postural drainage	Dead
613	26	M	Unknown	37	L. L.	Supportive	Unimproved
1463	45	F	Unknown	122	L. L.	Supportive	Dead
1632	47	M	Unknown	188	R. U.	External drainage	Dead
1652	60	M	Unknown	100	L. U.	Supportive	Dead
890	52	M	Unknown	110	R. U.	Supportive	Dead
3132	54	M	Appendectomy	2,924	L. L.	Supportive	Unimproved
5129	42	F	Unknown	70	R. L.	External drainage	Dead
4595	45	M	Unknown	51	L. U.	External drainage	Dead
4614	43	M	Tonsillectomy	22	L. U.	External drainage	Dead
3174	19	F	Dental extraction	215	L. L.	External drainage	Unimproved
2712	50	F	Unknown	122	R. L.	Supportive	Dead
3288	49	M	Unknown	355	R. L.	Supportive	Dead
4182	42	M	Unknown	119	R. U.	Postural drainage	Unimproved
1857	39	F	Unknown	83	R. U.	Supportive	Dead
1680	41	M	Unknown	134	R. U.	Postural drainage	Dead
564	50	M	Unknown	65	L. U.	Supportive	Dead
4046	38	F	Unknown	24	R. L.	Supportive	Dead
4385	34	M	Unknown	3,217	R. U.	Supportive	Unimproved
1985	36	M	Herniotomy	119	R. L.	Closed thoracotomy	Unimproved
2380	38	M	Unknown	791	L. L.	Supportive	Dead
5013	26	M	Unknown	167	R. L.	Postural drainage	Unimproved
4883	51	M	Aspiration (ammonia)	41	R. U.	Supportive	Dead
4180	44	M	Surgery of jaw	510	R. U.	External drainage	Dead
3527	55	M	Unknown	733	R. U.	Supportive	Dead
2854	37	M	Unknown	23	R. L.	External drainage	Dead
8240	47	M	Unknown	136	R. M.	Supportive	Improved
20645	20	F	Unknown	?	R. L.	Supportive	Unimproved
19629	65	M	Unknown	21	R. L.	Supportive	Dead
18799	57	M	Unknown	801	L. U.	Supportive	Unknown
13789	37	F	Unknown	79	R. L.	Postural drainage; ether-in-oil	Dead
10576	43	M	Unknown	76	L. U.	Supportive	Dead
10691	38	F	Tonsillectomy	241	L. L.	External drainage	Dead
12660	28	M	Unknown	459	L. L.	External drainage	Dead
170	43	M	Unknown	88	L. U.	Supportive	Unimproved
4199	77	F	Unknown	?	L. L.	Supportive	Dead
4285	63	M	Unknown	1,095	R. U.	Postural drainage	Unimproved
4106	66	M	Unknown	198	L. L.	Supportive	Dead
47331	55	M	Unknown	1,101	R. U.	Supportive	Dead
2273	65	F	Unknown	69	R. U.	Supportive	Dead
14560	35	M	Ulcerative tonsillitis	70	R. U.	Neosalvarsan	Cured
63740	36	M	Unknown	85	R. L.	External drainage	Cured
59157	45	F	Unknown	1,140	L. L.	Supportive	Unimproved
62922	55	M	Unknown	55	R. U.	Neosalvarsan	Unimproved
27931	40	M	Unknown	145	L. L.	Neosalvarsan	Improved
46738	48	M	Unknown	48	R. U.	Supportive	Dead
44903	46	M	Unknown	174	L. L.	Supportive	Unimproved
45109	35	F	Tonsillectomy	1,966	L. U.	Postural drainage	Unimproved
465	46	F	Unknown	19	R. U.	Supportive	Dead
50454	56	M	Unknown	2,920	L. L.	Supportive	Unimproved

TABLE V—CONT'D

CASE NUMBER	AGE	SEX	ETIOLOGY	DURATION (DAYS)	LOBE INVOLVED	TREATMENT	RESULT
49376	50	M	Unknown	415	R. L.	External drainage	Dead
49365	53	M	Unknown	107	R. U.	Supportive	Unimproved
49065	48	M	Unknown	123	R. L.	Bronchoscopic aspiration	Unimproved
31342	51	M	Unknown	574	L. L.	Supportive	Unimproved
52642	38	F	Unknown	67	L. U.	Supportive	Dead
51027	16	M	Tonsillectomy	415	L. L.	External drainage	Cured
48531	44	F	Hemiplegia	31	L. L.	Supportive	Dead
45208	44	M	Dental extraction	32	L. L.	Supportive	Unimproved
46864	40	M	Appendectomy	245	R. L.	Postural drainage	Cured
53654	25	M	Unknown	?	L. L.	Ether-in-oil; chest aspiration	Unimproved
58095	60	M	Unknown	213	L. L.	Neosalvarsan; KI; postural drainage	Unimproved
59437	18	F	Unknown	233	L. L.	Bronchoscopic aspiration	Unimproved
61113	60	F	Unknown	3,217	R. L.	Supportive	Dead
60424	42	M	Unknown	91	R. U.	Postural drainage; neosalvarsan	Unimproved
61737	2½	M	Tonsillectomy	33	R. L.	Supportive	Cured
64099	41	M	Submersion	148	L. U.	External drainage	Cured
69553	58	M	Unknown	290	R. L.	External drainage	Dead
79586	30	F	Unknown	603	R. L.	Supportive	Unimproved
81161	50	M	Unknown	82	L. L.	Sulfanilamide; guaiacol; postural drainage	Improved
64049	53	F	Unknown	120	R. L.	Supportive	Dead
63956	16	M	Unknown	133	R. L.	External drainage	Cured
71065	52	M	Unknown	279	R. L.	Postural drainage	Dead
57684	39	M	Unknown	157	R. U.	Supportive	Unimproved
73886	54	M	Unknown	821	R. L.	Supportive	Unimproved
73329	46	M	Unknown	141	R. U.	Supportive	Unimproved
30718	45	M	Nasal operation	32	R. L.	Supportive	Unimproved
67538	56	M	Unknown	154	R. U.	Postural drainage	Dead
67243	23	F	Mastoidectomy	1,829	R. L.	Supportive	Unimproved
63903	8	M	Unknown	661	R. L.	External drainage	Unimproved
66866	62	M	Unknown	389	R. U.	Supportive	Dead
72904	4	M	Tonsillectomy	301	L. U.	External drainage	Cured
69538	61	M	Unknown	51	R. U.	Supportive	Dead
70119	7	F	Unknown	57	R. L.	Postural drainage	Unimproved
73684	67	M	Unknown	198	R. U.	Postural drainage; guaiacol.	Unimproved
71613	52	M	Unknown	51	R. U.	Supportive	Dead
69012	47	M	Unknown	226	R. U.; R. M.	External drainage	Dead
69334	42	M	Unknown	158	L. U.	Postural drainage	Cured
71988	33	M	Unknown	82	L. L.	External drainage	Improved
20515	23	M	Aspiration of staple	3,650	L. L.	Removal of staple (Br.)	Cured
9540	48	M	Unknown	529	R. U.	Supportive	Dead
80879	59	M	Unknown	418	R. L.	Neosalvarsan	Dead
74327	50	F	Unknown	378	R. L.	Supportive	Dead
78092	32	M	Unknown	502	L. L.	External drainage	Dead
16765	61	M	Unknown	?	R. L.	Supportive	Unimproved

The treatment of putrid lung abscess is not a simple problem. There is a great deal of responsibility involved in deciding whether an abscess will heal spontaneously or will require operation. The successful treatment of the disease requires the joint cooperation of internist, bronchoscopist, roentgenologist, and surgeon.

SUMMARY

1. There were ninety-four cases of putrid lung abscess discharged from the City Hospital between the years 1920 and 1938, inclusive. The majority of patients were in the fourth, fifth, and sixth decades, and the ratio of males to females was 3 to 1.

2. Fifteen of the abscesses followed surgical procedures, of which two-thirds were of the tonsils, teeth, jaw, and nose. Seventy-nine were not preceded by surgery, and seventy-four had no predisposing condition whatsoever.

3. The pathogenesis and pathology of putrid abscess were described, and the anaerobic character of the infection was emphasized.

4. The incidence of abscess on the right side predominated over that on the left, but there was no marked difference in distribution between upper and lower lobes. The vast majority of abscesses was localized to a single lobe.

5. Putrid empyema was the most frequent complication and occurred in 12 per cent of the cases. The other important complications were cerebral metastases, pulmonary extensions, and bronchopneumonia.

6. The results of medical treatment were: Cured, 5 per cent; improved, 4 per cent; unimproved, 44 per cent; and dead, 47 per cent. The entire duration of illness in three-fourths of the deceased patients did not exceed one year.

7. Symptomatic improvement in the conservatively treated cases was a totally unreliable index of the ultimate prognosis; the discrepancies between roentgenologic and clinical findings were often marked; and recurrent exacerbations were frequent.

8. The results of surgical treatment were: Cured and improved, 29 per cent; unimproved, 14 per cent; dead, 57 per cent. The majority of deaths were either operative or accidental, and the high mortality rate does not accurately reflect the effect of surgery on putrid abscess.

9. The various medical procedures used in the treatment of putrid lung abscess have no definite therapeutic value. Whatever improvement has resulted with conservative measures has been spontaneous and independent of specific treatment.

10. The poor results of conservative treatment are obvious indictments of this form of therapy. The type of infection and the pathologic changes present in this disease make it apparent that early ex-

cision and aeration of the abscess is the rational solution to the problem. Where this procedure has been followed, the percentage of cures is maximal and the mortality rate negligible.

REFERENCES

1. Cutler, E., and Gross, R.: Nontuberculous Abscess of the Lung, J. THORACIC SURG. **6**: 125, 1936.
2. Flick, J., Clerf, L., Funk, E., and Farrell, J.: Pulmonary Abscess, Arch. Surg. **19**: 1292, 1929.
3. Neuhof, H., and Touroff, A.: Acute Putrid Abscess of the Lung, Surg. Gynec., & Obst. **66**: 836, 1938.
4. Stern, L.: Etiologic Factors in the Pathogenesis of Putrid Abscess of the Lung, J. THORACIC SURG. **6**: 202, 1936.
5. Neuhof, H., and Wessler, H.: Putrid Lung Abscess, J. THORACIC SURG. **1**: 637, 1932.
6. Bucher, C.: Abscess of the Lung, Am. J. Med. Sc. **179**: 406, 1930.
7. Cohen, J.: The Bacteriology of Abscess of the Lung and Methods for Its Study, Arch. Surg. **24**: 171, 1932.
8. Weiss, C., and Mercado, D.: Studies of Anaerobic Streptococci From Pulmonary Abscesses, J. Infect. Dis. **62**: 181, 1938.
9. Allan, C., and Blackman, J.: Treatment of Lung Abscess, J. THORACIC SURG. **6**: 156, 1936.
10. Brunn, H.: Lung Abscess, J. A. M. A. **103**: 1999, 1934.
11. Lord, F. T.: Diseases of the Bronchi, Lungs and Pleura, Ed. 2, Philadelphia, 1925, Lea and Febiger, p. 460.

45 GRAMERCY PARK NORTH

THE LOGIC OF COLLAPSE THERAPY

A Mathematical, Physiologic, and Clinical Study

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IT IS strange that physicians and surgeons, although they are representatives of an important branch of natural science, do not care very much about mathematical problems. Philosophers do. When Immanuel Kant,[1] in 1763, tried to introduce the "conceptions of negative values into the world's wisdom," he was guided, originally, by mathematical principles: Plus is plus and minus is minus. In the plane as well as in space, plus moves in one direction and minus in the opposite direction. If we add plus to plus, we get plus. If we add minus to minus, we get minus. And if we add plus to minus, equally distant from the zero level, the result is zero. Kant's principles apply, not only figuratively, but also actually, to our problems of collapse therapy in many respects. The difficulties in establishing a plan for the proper management of a case of pulmonary tuberculosis, the yes and no of our conflicting concepts in handling a case of cavernous tuberculosis complicated by a severe extrapulmonary lesion, the "approach to the organism as a whole"[2] which demands a conservative management in cases that would be suitable for collapse therapy if the lesion would be confined to the lung alone, the decision about the type of purely mechanical procedures in localized pulmonary lesions, the yes and no of discontinuance of artificial pneumothorax, the final verdict as to the approach of a residual cavity following thoracoplasty in an otherwise healthy person—all these considerations demand our utmost vigilance. Here is an example: In one of the leading sanatoriums in the East a girl was ready to be discharged after a three-stage thoracoplasty when the roentgenologist stated in his final report that there was a suspicious highlight visible in the collapsed area. The sputum occasionally showed one tubercle bacillus per field. From the clinical viewpoint the patient was practically recovered. The final sentence of the medical committee, however, after serious consideration of the pro and con, was a revision of the operation, with complete closure of the residual potential cavity in view. The patient died of a tuberculous meningitis soon after the operation. Was the verdict of the committee right or wrong? From the purely mechanical "viewpoint" (sensual "Anschauen"[7]), they were right. From the viewpoint of the physician who should make his decision

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by looking at the person as a whole, they were wrong. The patient, without a further operation, would probably have lived for many years, if not to a normal age.

Another patient with a cavernous left lung, successfully collapsed by thoracoplasty, developed a small parahilar cavity on the right which demanded, from the mechanical viewpoint, some form of extensive collapse therapy with a sacrifice of normal breathing space. An artificial pneumothorax proved successful. The cavity was closed, but the patient suffered from severe dyspnea. Without pneumothorax on the right he would have had a chance to live quite comfortably for a few years. With pneumothorax, however, he was mechanically "cured," although his milieu was extremely limited. He was doomed to invalidism through lack of breathing space and to a slow death from right heart failure. Every narrowing of the patient's milieu has a tremendous influence upon his organism as well as upon his character. The physician, therefore, before he decides upon the mechanical collapse of the so-called better lung in a case like the one described, will have to consider whether or not, with the treatment suggested, he handicaps the patient's chances beyond his power of adjustment. Kurt Goldstein,[2] in his "wholistic" approach of the patient as an individual, is right in saying, "Every irreparable change of the organism limits its very nature and so its responsiveness if the latter is not restored in some other way." Gradual discontinuance of the pneumothorax on the good side will, therefore, be the logical thing in this case.

The main problem in mechanical phthisiotherapy is the anatomic closure of the cavity. The fate of the patient depends upon it. If the collapse is effective, the patient will get well. If it is ineffective, we must admit a failure. Collapse treatment in a noncavernous tuberculosis is illogical. Infiltrative "changes" due to a tuberculous infection as such do not justify a mechanical approach. If central necrosis and caseation are absent, then such changes will become absorbed spontaneously in the great majority of the patients if they are put at rest and properly watched with x-ray examinations. These "changes" alone, therefore, do not actually mean disease in the surgical sense of the word. "Only when a change begins to become a danger [i.e., cavity], then it becomes a disease."[2]

When Graham,[14] at a meeting in Washington, criticized the paper of a young surgeon who tried to prove that a pulmonary abscess is curable by removing a small piece of a rib and pushing the diseased part of the lung inward with a gauze pack, the audience readily understood the reason for Graham's remarks. A pulmonary abscess as we know it today is caused mainly by anaerobes. An attempt, therefore, simply to compress air-containing lung tissue around the abscess would be illogical. Obviously anaerobes would grow in compressed,

atelectatic lung tissue much faster than in a normal air-inflated lung. For the same reason, a compression of lung tissue around the abscess by insufflation of air into the pleural sac under positive pressure would be illogical. The only acceptable pneumothorax treatment in lung abscesses, under exceptional conditions, would be the insufflation of smallest doses of air given at short intervals under an inspiratory and expiratory pressure negative enough to avoid atelectasis around an abscess localized centrally, i.e., close to the mediastinum. As Harrell states, "The objective is to promote and facilitate drainage, as it is through the process of drainage that pyogenic infections are thrown off. . . . Small quantities of air should be injected into the pleural cavity to act as a buffer and to prevent the lung from sticking. The wavelike motion through inspiration and expiration will continue, and the lung will be in a better position to empty itself."[6] Harrell's suggestions would be illogical, of course, in lung abscesses localized just under the surface of the visceral pleura adherent to the chest wall. An attempt to insufflate air in such instances would be not only illogical, but dangerous, on account of the possibilities of a tear in the lung and subsequent empyema. Furthermore, any future surgical approach would entail preliminary operation to establish such adhesions. The frontal and lateral Bucky film in such cases will help to localize the abscess and guide our therapeutic procedures.

Another illogical attempt to obliterate a cavity, for instance a tuberculous cavity, is the method of Neuhof, called by him "pneumocavernolysis." Neuhof advises removal only of those parts of the ribs which cover the tuberculous cavity and "compression" of the cavity with a gauze pack for six weeks. His final results, published by Potter,[3] were unsatisfactory in a large number of cases. This unfavorable outcome is explained by the fact that the average cavity treated by collapse therapy requires at least eighteen months to guarantee a permanent anatomic closure. In other words, the healing of such a cavity cannot be accomplished by a six weeks' "compression," but must be done by a logical collapse, which means by an artificial support of the lung in its tendency to "return to its embryonic state."[4]

The embryologists have taught us that the lung of an embryo lies collapsed in the costovertebral gutter in a completely atelectatic state. The space between the surface of the collapsed lung of the embryo and the inner surface of the chest wall shows a zero pressure originally. After the first respirations the lungs expand gradually and until the two pleural layers meet. In other words, the expansion of the lungs in the newborn infant is governed mainly by one factor, the intrabronchial atmospheric pressure. The question of what second factor keeps the lung expanded and prevents it from its natural tendency to retract into the costovertebral gutter is still undecided. Some authors believe that the rapid growth of the infant's chest wall is the

cause of the potential negative intrapleural pressure which, in connection with the physiologic adherence between the two pleural layers, holds the lungs in expansion. The writer is inclined to agree with Henry Sewall,[11] who says: "Cohesion between the pleural surfaces is strong enough to withstand the elastic pull of the lung. This is proved by placing the thorax of an animal within the jar of an air pump; the lung continues in contact with the chest wall after exhaustion of the air."

If one introduces a needle into the pleural sac, the manometer shows a negative reading as soon as the air present in the needle and in the connecting rubber tube has entered the pleural vacuum. This proves the natural tendency of the lung to retract. Another proof is the interesting phenomenon which one notices as soon as one begins to unroof the diseased part of the lung by thoracoplasty. A paradoxical movement of the lung, a "sinking inward," takes place during inspiration. This again indicates that collapse, and not compression, is the thing that actually takes place when we attempt to obliterate the tuberculous cavity. With due respect to Lilienthal, one must state that his conception of lung compression is wrong and the term ill-chosen, and that collapse is the only acceptable explanation. The same objection applies to Sauerbruch's standard thoracoplasty. His "brusteinengende" (narrowing the chest) operation, by which he removes only small pieces of eleven ribs, not only fails to close the apical cavity but also compresses the lower parts of the lung. In this manner he sacrifices normally functioning lung tissue which, especially in bilateral selective apical collapse, should be saved as a necessary breathing space. Sauerbruch's standard approach is, therefore, illogical and is today of historical value only.

The spontaneous so-called Coryllos closure of the cavity following blockage of the draining bronchus and subsequent atelectasis of the diseased portion of the lung, although doubted by M. Pinner[16] and others, must be accepted as a proved logical phenomenon.[15] If the draining bronchus is blocked, the intracavernous air becomes gradually absorbed and the cavity closes (adding "minus to minus"), provided, however, that the pericavernous tissue is retractable and does not interfere with the closure. In partially blocked bronchi, on the other hand, in which air enters the cavity without being able to escape (adding "plus to plus"), the cavity becomes larger. There is one thing, however, which the Coryllos school does not seem to consider sufficiently, namely that a reopening of the cavity takes place as soon as the bronchial passage is free, for instance, after expectoration of the blocking plug. A permanent Coryllos closure of the cavity seems to be logical only if one should succeed in obliterating the draining bronchus permanently by mechanical means. Such attempts have

proved to be failures until now. Furthermore, one must realize that bacteria, anaerobes, for instance, trapped behind the bronchial closure, could cause a severe infection leading to pneumonitis and its consequences.

The so-called giant cavity deserves special attention. Not every large cavity is a giant cavity. The writer understands a giant cavity to be a hole occupying an entire lobe with pericavernous lung tissue completely absent, its wall consisting of a pleural shell only. It is impossible to conceive how such a cavity could ever close spontaneously in the Coryllos manner. There is no retractable lung tissue; the pleural shell is adherent to the cage. The cavity can neither close in the purse-string manner (Ulmar[12]) nor by forced approximation of its walls. It is, therefore, not surprising that until now giant cavities have failed to respond to collapse therapy. The future attempts to approach giant cavities will be either cavernostomies or lobectomies.

The high-pressure pneumothorax suggested by Leuret and Delmas-Marsalet and others with a view towards compression of the lung and given at short intervals under an initial and final positive pressure must be condemned as illogical. An attempt to stretch pleural adhesions by "hammering" under positive intrapleural pressure not only causes a shift of the mediastinum to the opposite side, but endangers the life of the patient by a possible rupture of the cavity localized just under the surface of the lung and held open by a pleural band. The only logical treatment of such a case is the electrosurgical severance of pleural adhesions with subsequent pneumothorax refills given under negative pressure at short intervals. Thus, one supports the lung in its natural tendency to retract and, at the same time, allows the mediastinum, under fluoroscopic control, to remain in the midline position. But even if there are no pleural adhesions interfering with the collapse, one is often tempted to "hammer" the lung under positive pressure in cases of cavities with a stiff wall, for example, or in parahilar cavities which, as we know, respond very rarely, and then with difficulty, to collapse therapy. Here, too, a mediastinal hernia, especially in the upper part of the mediastinum, develops and interferes with the proper retraction of the diseased lung. The logical attempt to close such a cavity is to fix the mediastinum, first, as suggested by Potter[10] and supported by Baum,[9] by an artificial effusion following injection of sterile oil into the pleural sac, and then to continue easily with refills given at short intervals under negative pressure. A proper midline position of the mediastinum is, in such cases, most essential. This can be obtained by frequent fluoroscopic controls. It would be illogical, of course, to fix a herniated mediastinum. The cavity would never close after malfixation. In other words, the lung is able to retract in its own hemithorax only.

The formation of fluid in the pleural sac in artificial pneumothoraces is a much discussed problem. The etiological factor is not always clear. Ulmar's[12] explanation that an antigenic reaction occurs on the pleural surface is acceptable in those cases in which the exudative character of the disease still prevails, but his conception that intrapleural pressure has "very little bearing on the formation of fluid" is erroneous. Graham's[13] animal experiments, as well as our clinical observations in numerous instances, have proved beyond doubt that pressure changes from high negative to high positive play a very important part in the formation of pleural fluid. This is especially noticeable in pneumothoraces of long duration given at long intervals. The intrapleural pressure in those cases is often so strongly negative that, after insertion of the needle, air enters the pleural sac with an audible rush. Similarly, fluid formation is almost a rule in cases in which pneumothorax treatment is discontinued. A temporary phrenic nerve interruption in such cases, with a narrowing of the pleural space in view, sometimes prevents the formation of fluid.

Until about two years ago there existed a great deal of confusion about phrenic nerve surgery in cavernous tuberculosis. The statistics (7 per cent of cavity closures on one hand and 90 per cent on the other) were so contradictory that some strict rules about phrenic nerve interruption were necessary. The indications of B. P. Potter, Berry, and Bortone[5] seem to be the most logical ones. According to these authors, one can expect an obliteration of a cavity smaller than an inch or less in diameter if the cavernous wall is soft enough and if an appreciable amount of pericavernous elastic lung tissue is present to guarantee retraction. Even in cases of pericavernous infiltration one can expect a closure if films taken at short intervals following phrenic nerve interruption reveal gradual absorption of the infiltrated area and a tendency of the affected lobe to retract. A logical procedure is, furthermore, phrenic nerve surgery supplementing an artificial pneumothorax ineffective on account of apical and diaphragmatic adhesions. It is contraindicated, however, in those cases in which the failure of the cavity to close is due to a lateral adhesion, in cavities adherent to the chest wall at its outer border, in cavities covered with a thick pleural cap, and in multiple and large cavities.[5]

Extrapleural apicolysis with plombage is not very popular in this country. The reason is obvious. The diseased lobe is not unroofed, but only separated by cleavage from the chest wall with the finger or a blunt instrument following resection of a small part of the third or fourth rib posteriorly. A retraction of the lobe and cavity closure can be expected only if the cleavage is thorough enough and if a foreign body material is introduced extrapleurally in order to keep the lobe separated permanently from the chest wall. The type of

material used in plombage is irrelevant, provided the foreign body does not irritate. Paraffin has been used extensively. Instances of foreign body reaction with subsequent perforation into the apical cavity and expectoration of the paraffin were published by many au-

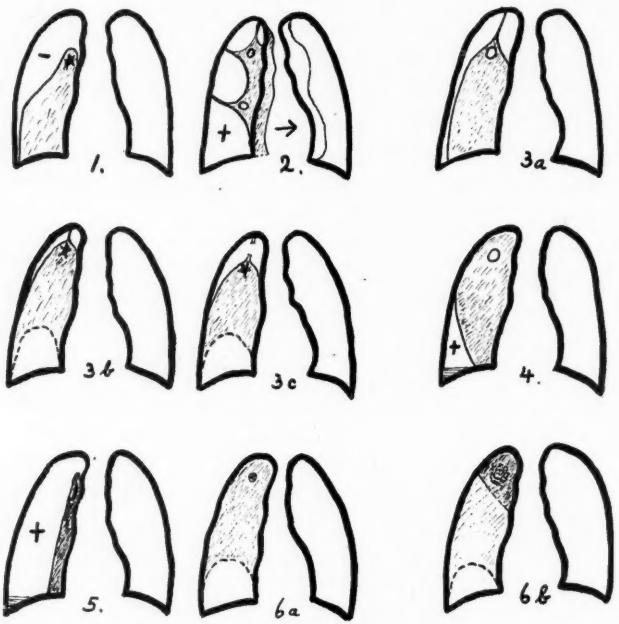

Fig. 1.—*1.* Ideal selective pneumothorax. Diseased upper lobe retracted; lower lobe expanded; cavity closed. Intrapleural pressure negative before and after refills.

2. High pressure pneumothorax. Intrapleural pressure positive before and after refills. Mediastinum and heart shifted to the left. Mediastinal hernia. This is an illogical procedure. In our attempt to stretch pleural adhesions by high positive pressure we endanger the patient by a possible rupture of the cavity held open by adhesions. The only logical approach is the severance of the adhesions and continuance of the pneumothorax with small doses of air given at short intervals under negative pressure. An ideal selective pneumothorax would be the result.

3a. Ineffective pneumothorax due to apical and diaphragmatic adhesions.

3b. Supplementary phrenic nerve interruption. The rising diaphragm relaxes the apical adhesion, permitting the upper lobe to retract and the cavity to close.

3c. Electrosurgical severance of the apical adhesion after phrenic nerve interruption had proved to be insufficient.

4. Illogical attempt to collapse an apical cavity by contraselective pneumothorax. Air pocket collapsing only the healthy lower part of the lung. Result frequently an empyema. Discontinuance of pneumothorax and surgical collapse advisable.

5. Illogical artificial pneumothorax as practiced in the early days. Intrapleural pressure is positive. Unnecessary collapse of healthy lung tissue accompanies that of the cavernous apex. This frequently results in complete atelectasis or inability of the lung imprisoned by the thick visceral pleura to re-expand. In such cases the pneumothorax must either be continued indefinitely or the cage of the affected side removed surgically.

6a. Logical collapse by phrenic nerve interruption. Cavity small or medium in size. Pericavernous infiltration absent.[5]

6b. Phrenic nerve interruption. Cavity within retracting upper lobe failed to close anatomically. Pericavernous infiltration present but not very pronounced. Cavity had diminished in size. In such cases fluoroscopic control is essential and immediate reinterruption advisable if diaphragm begins moving again before cavity is closed completely. But even in case of cavity closure, temporary or permanent reinterruption will often be necessary.

thors. The writer has used hydrostearolein, known as Crisco, in several cases, with variable results.* Unfavorable effects such as those in paraffin plombage were never noticed. The operation has been abandoned lately in favor of extrapleural pneumothorax.

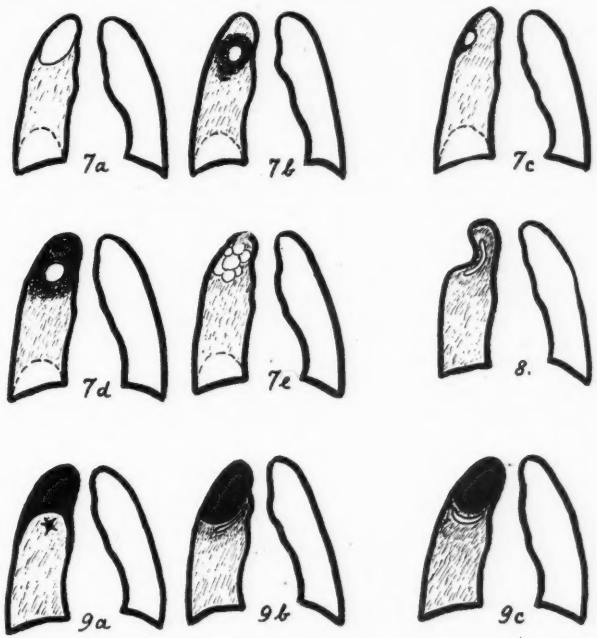

Fig. 2.—7*a*. Illogical phrenic nerve interruption in the case of a huge cavity in upper field adhering to chest wall. No retractable pericavernous lung tissue, but a pleural shell only.

7*b*. Illogical phrenic nerve interruption. Cavity with thick fibrous wall surrounded by fibrous and fibrocavernous infiltration.

7*c*. Illogical phrenic nerve interruption. Cavity localized peripherally close to the outer border of the chest wall and adherent to same.

7*d*. Cavity surrounded by thick pleural tissue. Phrenic nerve interruption illogical. Lobe unable to retract.

7*e*. Multiple cavities do not respond to phrenic nerve interruption. According to Potter and co-workers,[5] they not only do not close but combine into a large antrum. Retraction of lobe impossible for similar reasons.

8. Illogical pneumocavernolysis (Neuhof). Parts of two ribs removed; cavity pushed downward. Re-expansion of lung and reopening of cavity in a great number of cases after removal of gauze pack.

9*a*. Plombage following apicolysis acceptable only if subfascial cleavage extensive enough to guarantee sufficient retraction of lobe and cavity closure.

9*b*. Plombage. Insufficient cleavage. Unacceptable.

9*c*. Plombage. Insufficient cleavage. Unacceptable.

Extrapleural pneumothorax following subfascial pleurolysis is still in the experimental stage. It is safe to state today that it will never replace selective thoracoplasty. High pressure refills are illogical

*The writer is indebted to Procter and Gamble for furnishing sufficient material of hydrostearolein specially prepared with a melting point of 125° F. Animal experiments with reference to the foreign body reaction and harmlessness of hydrostearolein were performed in collaboration with Dr. S. A. Goldberg, Director of Laboratories, Presbyterian Hospital, Newark, N. J.

both intrapleurally and extrapleurally. However, as a lifesaving procedure, this operation may be acceptable in cases where thoracoplasty would be too great a risk. A permanent result can be expected only in rare instances. It will probably hold its place as a preliminary operation to thoracoplasty, although the dangers of infection following such subsequent surgery should be borne in mind.

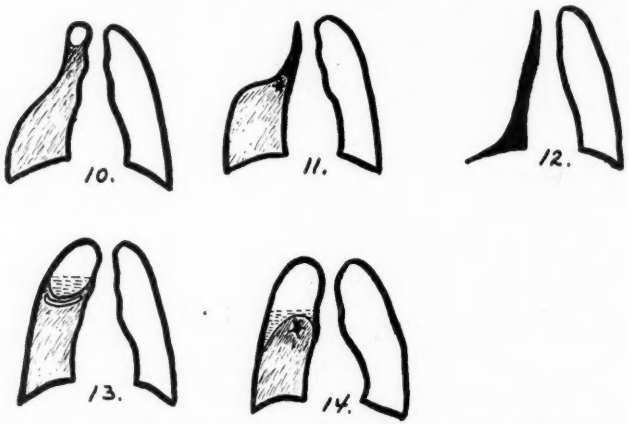

Fig. 3.—*10.* Illogical ineffective thoracoplasty. Stump of first rib too long. Cavity, localized just below stump and adherent to same, fails to close.

11. Ideal upper lobe thoracoplasty. Selective collapse. Cavity closed. Lower lung field expanded and functioning.

12. Illogical old standard thoracoplasty. Apical cavity closed, but healthy lower lung field unnecessarily collapsed and its function sacrificed.

13. Illogical extrapleural pneumothorax following subfascial pleurolysis. Cavity walls approximated but cross union not imaginable on account of insufficient cleavage.

14. Extrapleural pneumothorax after sufficient cleavage. Cavity closed, but permanent result questionable. An upper stage thoracoplasty will be necessary.

SUMMARY

An attempt has been made to apply the laws of logic and physiology to our present-day conception of collapse therapy.

Compression of a lung under negative pressure is a nonsensical contradiction. Collapse under negative pressure is logical. Collapse under positive pressure is illogical. The only logical procedure in collapse therapy is to support the structures surrounding the diseased part of the lung in their natural tendency to retract.

REFERENCES

1. Kant, Immanuel: Kritik der reinen Vernunft.
2. Goldstein, Kurt: Die ganzheitliche Betrachtung in der Medizin (The "Wholistic" Approach of the Organism in Medicine), Kongress zur Förderung medizinischer Synthese und ärztlicher Weltanschauung, Leipzig, 1933, Th. Steinkopff, p. 155.
3. Potter, B. P.: J. THORACIC SURG. **4**: 288, 1935.
4. Rost-Reimann: Pathological Physiology, p. 372.
5. Potter, Berry, and Bortone: J. THORACIC SURG. **6**: 424, 1938.
6. Harrell: Virginia M. Monthly **63**: 134, 1936; J. A. M. A. **107**: 541, 1936.

7. Husserl, Edmund: Logische Untersuchungen, Halle, 1922, Max Niemeyer, Vol. II, p. 201.
8. Baum, F.: Radiology **25**: 730, 1935.
9. Baum, F.: Radiology **27**: 359, 1936.
10. Potter, B. P.: J. THORACIC SURG. **7**: 382, 1938.
11. Sewall, H.: Am. Rev. Tubere. **18**: 128, 1928.
12. Ulmar, D.: Tuberculosis Conference, Cornell University, X. 7, 1936; Quart. Bull., Sea View Hosp. **2**: 307 and 425, 1937.
13. Graham, E. A.: Surgical Diseases of the Chest, Philadelphia, 1935, Lea and Febiger, p. 19.
14. Graham, E. A.: Discussion, Meeting American Association for Thoracic Surgery, Washington, D. C., 1933.
15. Coryllos and Ornstein: J. THORACIC SURG. **8**: 26, 1938.
16. Pinner, M.: Ann. Int. Med. **9**: 513, 1935.

PRIMARY (BRONCHIOGENIC) CARCINOMA OF THE LUNG

With a Histopathologic Study of Forty Necropsies

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AN EXTENSIVE and detailed review of the literature on the pathology of bronchiogenic carcinoma is precluded in this paper, so that only a generalized summary of the prevailing conception will be given. Malignant neoplasms of the lung may arise either from the epithelium lining the bronchi, from the epithelium of the bronchial mucous glands, or from the alveolar epithelium. Tumors belonging to the latter group will not be considered in this communication. Carcinoma arising from the bronchial epithelium has been said to give rise to many histologic varieties, but no definite attempt has been made heretofore to classify them adequately, particularly with regard to their cell morphology. Few tumors, indeed, present a more variable and complicated histology than those of bronchial origin, and there is still much discussion as to their histogenesis. These tumors often present a multiplicity of cell appearance and cell structure, which bear little, if any, resemblance to the parent tissue from which they are known to arise. On the other hand, tumors which arise from the bronchial mucous glands are said to present a very characteristic microscopic picture. Briefly, they mimic very closely the parent tissue in so far as their cell structure and tissue pattern are concerned. The cells assume a glandular formation, are cylindrical or cuboidal in shape, and often continue with their function of mucus production.

In general, it may be said that cancer arising from the epithelium lining the bronchus presents a large bulky growth which advances along its wall, often invading it, leading eventually to ulceration, obstruction, secondary infection, and even to perforation. Carcinoma of mucous gland origin, on the other hand, is chiefly limited to the wall of the bronchus from which it arises and usually produces a diffuse infiltration and thickening of the bronchial wall which ultimately causes stenosis of the bronchus. This type of tumor is confined, as a rule, to the submucosa while the epithelial lining is spared and remains intact. The former type of neoplasm is apparently less malignant than tumors belonging to the latter group. In order to

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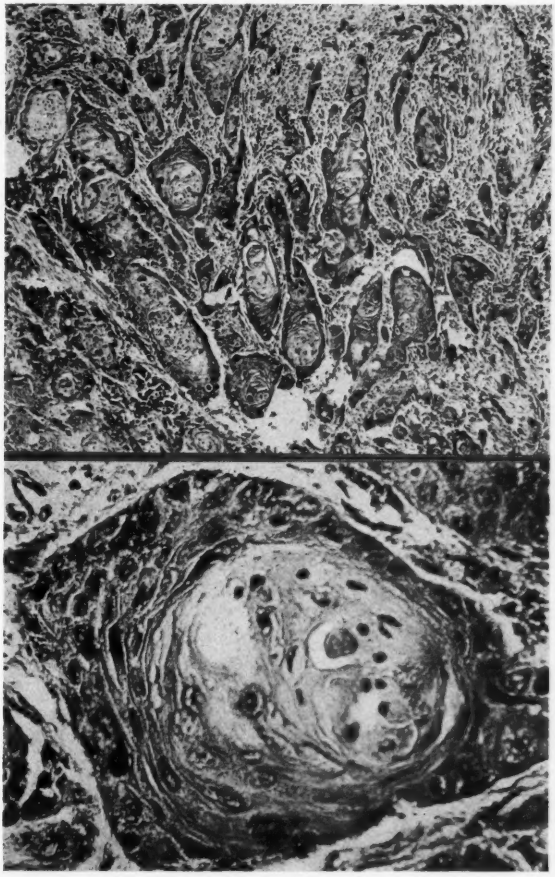
obtain further information regarding these tumors, we have reviewed and are reporting this group of forty necropsies. Furthermore, a discussion will be presented of the histology and histogenesis of carcinoma of the lung as it has been noted in this study. The cases under review have been grouped according to the histologic picture presented by the various tumors.



A.

B.

Fig. 1*A*.—Squamous cell carcinoma with typical pearl formation. (×100.)
Fig. 1*B*.—Typical epithelial pearl in a squamous cell carcinoma. Note the prickle cells. (×700.)

Of the forty cases of carcinoma of the lungs, twenty-three (57.5 per cent) had their origin in the right bronchial tree, and seventeen (42.5 per cent) in the left. The main bronchi were the seat of the growth in fifteen, or 37.5 per cent, of the cases. Of the twenty-three right bronchus tumors, seven arose from the main bronchus, whereas, of the

seventeen left bronchus tumors, eight arose from the main bronchus. The upper lobe bronchi were involved eleven times on the right and six times on the left side (Table I). These findings are more or less in accord with those reported in the literature and require no further comment. The gross characteristics of the various tumors in our

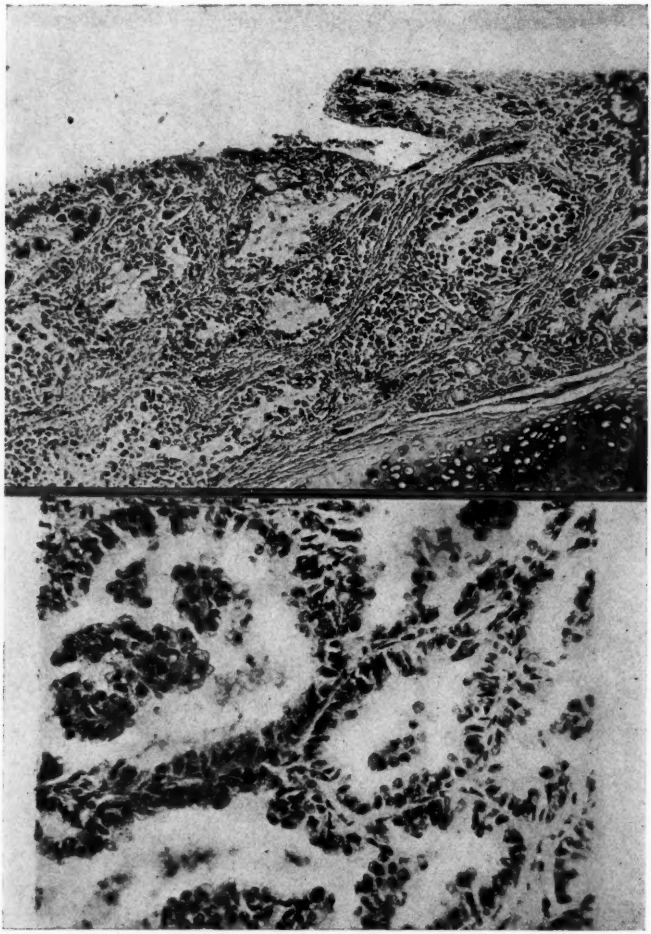


Fig. 2*A*.—Columnar cell (adeno)carcinoma. (×100.)
Fig. 2*B*.—Adenocarcinoma showing glandular lumina lined by tall cylindric cells. (×460.)

series presented no special features. Some of the tumors were small and localized to the bronchus from which they arose, while others were large and bulky and extended into the lung parenchyma. Some

showed widespread metastases to adjacent and distant organs; the latter represented the most malignant forms of growth.

TABLE I

SITES OF ORIGIN IN FORTY CASES OF BRONCHIOGENIC CARCINOMA

LOCATION	RIGHT SIDE	LEFT SIDE	TOTAL
Main bronchi	7	8*	15
Upper lobe bronchi	11	6	17
Lower lobe bronchi	2	3	5
Middle lobe bronchus	1		1
Middle and upper lobe bronchus	1		1
Middle and lower lobe bronchus	1		1
Total	23	17	40

*Two cases in this series began to the left of the carina and extended partly into the right main bronchus.

Of particular interest are the histologic characteristics presented by the various tumors. Many showed such wide morphologic variations that they bore no cytologic resemblance whatsoever to the

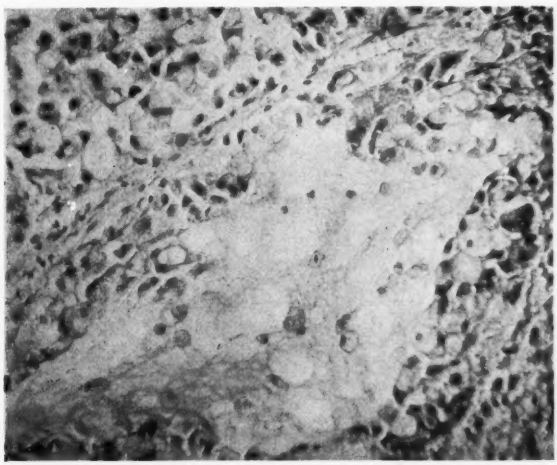


Fig. 2C.—Adenocarcinoma with mucus-secreting cells. (×460.)

bronchial epithelium from which they originated, while others mimicked the parent tissue very closely. Based upon their morphologic characteristics alone, these neoplasms fell into two distinct cytologic forms, namely, (1) squamous cell carcinoma and (2) cylindric cell carcinoma. A third group may be added, namely, those in which the tumor presented more than one cell type variety. It has been elected to call the latter group (for the present) "pleomorphic cell carcinoma" because of the many different forms of cells composing the neoplastic process. Fried has included the latter group under the term of bronchiogenic basal cell carcinomas.

CLASSIFICATION OF BRONCHIOGENIC CARCINOMA

1. Squamous cell carcinoma
2. Cylindric cell carcinoma
   A. Adenocarcinoma
   B. Undifferentiated cell carcinoma
      a. Medullary carcinoma
      b. Round cell carcinoma
      c. Oat cell carcinoma
3. Pleomorphic cell carcinoma

The *squamous cell carcinoma* formed only 10 per cent (4 cases) of this series. This figure is rather low as compared with other figures of similarly reported types of bronchial carcinoma. The apparent discrepancy in results between the low figures in this series and the higher figures reported by others in the literature may be explained on the basis of differences in interpretation of the histologic picture of the lesion. We feel that certain essential criteria are necessary to fulfill the requirements for the diagnosis of squamous cell carcinoma, the most important of which is the presence of prickle cells and the formation of the typical pearl. It seems that many cases have been included under the heading of squamous cell carcinoma when, in reality, they are transitional phases in the development of the various forms of cylindric cell carcinoma. The squamous cell carcinoma cases reported in this paper showed prickling of the cells and typical pearl formation so characteristic of this group of neoplasms (Figs. 1*A* and 1*B*).

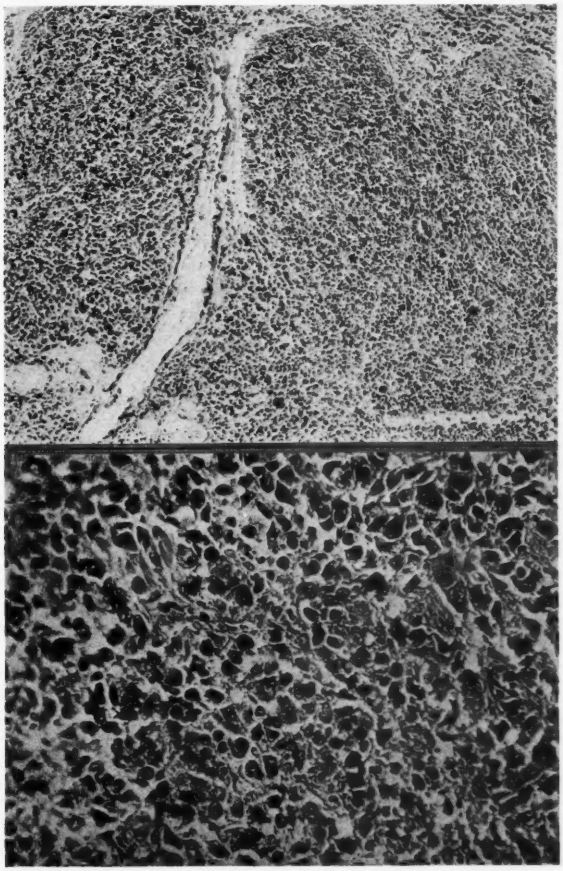
There were thirty-one cases of *cylindric cell carcinoma* in this series (77.5 per cent). These cases could be differentiated further according to their cell structure into (1) adenocarcinoma and (2) undifferentiated cell carcinoma. The adenocarcinomatous tumors numbered fourteen (35 per cent) of this series, whereas the undifferentiated tumors numbered seventeen (42.5 per cent) of the cases.

The striking feature of the *adenocarcinoma* group was the formation of glandlike structures which closely resembled the bronchial mucus glands. The cells were tall, columnar in shape, surrounding a central lumen, and were often found to contain mucus in the cell cytoplasm itself or in the lumen of the gland (Figs. 2*A*, 2*B*, and 2*C*).

The *undifferentiated tumors* failed to show any resemblance whatsoever to the bronchial epithelium from which they developed. They showed three different, but distinct, types of cells and were classified accordingly as medullary carcinoma (three cases), round cell carcinoma (twelve cases), and oat cell carcinoma (two cases).

The *medullary cell carcinoma* was characterized by the presence of tall columnar or polygonal cells arranged in nests and strands closely packed together, and it showed no tendency toward organization or definite pattern formation (Figs. 3*A* and 3*B*).

The cells composing the *round cell* form of undifferentiated cylindric cell carcinoma showed a very striking resemblance to the small lymphocyte of the circulating blood. The cells were small and round, of about the same size and shape as a blood lymphocyte. The nucleus was also round, hyperchromatic, and occupied the major portion of the cell substance, while the cytoplasm was scant and formed but a narrow rim surrounding the periphery of the nucleus. The structural arrangement



A.

B.

Fig. 3*A*.—Medullary carcinoma showing a diffuse growth of undifferentiated cylindric cells. (×100.)

Fig. 3*B*.—Medullary carcinoma in which the cells are tall, undifferentiated, and contain hyperchromatic nuclei. (×460.)

of these cells varied in different tumors and sometimes in different sections of the same tumor. In places the cells were found crowded together into dense nests with very little intercellular connective tissue stroma to support them; in others, they assumed a papillary-like formation, the cells being mounted in single or multiple rows upon a thin stalk of fibrous connective tissue (Fig. 4).

The *oat cell carcinoma* was so termed because cytologically it simulated oat seeds. These cells were elongated, oval, or spindle-shaped, arranged in columns, and resembled sheaves of oats packed together. Here again, the cell cytoplasm was scant while the nucleus was relatively large and occupied the major portion of the cell. There was very little

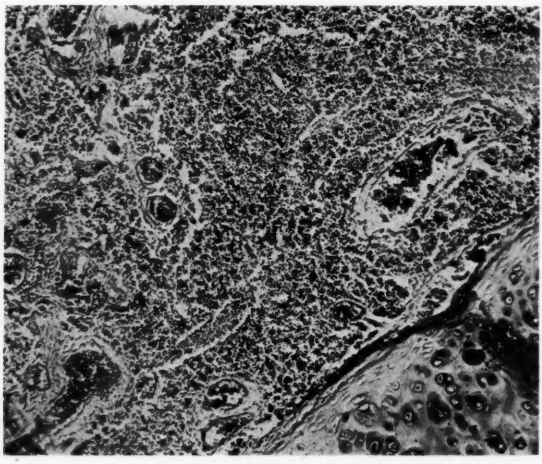


Fig. 4.—Round cell carcinoma. The cells are small, round, and arranged in sheets without tendency toward organization. (×100.)

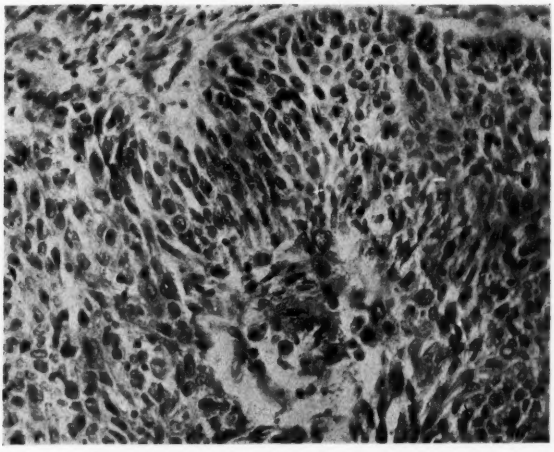


Fig. 5.—Oat cell type of carcinoma. The cells appear spindle-shaped and are arranged in densely packed nests. (×460.)

connective tissue stroma to be found in these tumors. The cell pattern varied somewhat in different sections of the tumor, being papillary-like in some areas, while in others it was arranged in columns, sheets, and nests of cells (Fig. 5).

The *pleomorphic* group numbered five (12.5 per cent) of the tumors. They displayed the various combinations of cell morphology described above. Thus, two cases showed a combination of adenocarcinoma and squamous cell carcinoma in the same tumor (Fig. 6); one case showed a combination of adenocarcinoma, round cell and oat cell carcinoma;

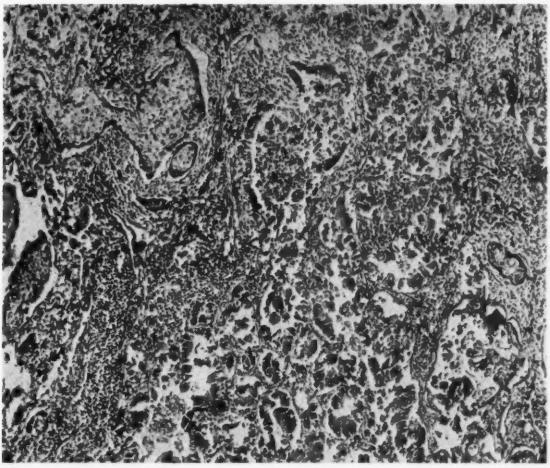


Fig. 6.—Pleomorphic cell carcinoma with squamous and adenocarcinomatous forms. (×100.)

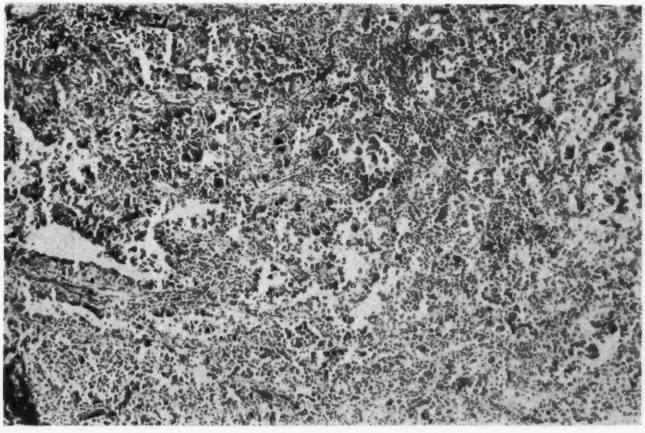


Fig. 7.—Pleomorphic cell carcinoma. There is a combination of round, oat, and cylindric forms of cells. Also note the large multinuclear giant cells. (×100.)

one, adenocarcinoma and round cell carcinoma; and one, a combination of medullary and oat cell carcinoma. These tumors also showed, in addition to either the cylindric or squamous cells, large, irregularly

TABLE II

COMPOSITE ANALYSIS OF METASTATIC INVOLVEMENT IN FORTY NECROPSIES FOR BRONCHIOGENIC CARCINOMA

SITE	NUMBER	SQUAMOUS CELL	ADENOCARCINOMA	MEDULLARY	ROUND CELL	OAT CELL	PLEOMORPHIC
Head and Neck							
Cervical lymph nodes	6		2	2			2
Brain*	6		4		1		1
Thyroid	4		2	1			1
Skull	1		1				
Pituitary	1		1				
Jugular vein	1				1		
Clavicle	1		1				
Total number of deposits	20	0	11	3	2	0	4
Thorax							
Tracheobronchial lymph nodes	34	4	13	2	10	1	4
Same lung	12	2	3		6		1
Ribs	10	1	4	1	3		1
Both lungs	10		6	2	1		1
Mediastinum	8	1	3		2	1	1
Pleura on same side	7		2		2	2	1
Trachea	6	1	1	1	2		1
Pericardium	5	1	3				1
Pulmonary vein	5	1		2	1	1	
Diaphragm	3	1	1		1		
Heart	3		3				
Opposite lung	3		1		1		1
Superior vena cava	2		1		1		
Esophagus	2		1				1
Pleura on both sides	1		1				
Axillary lymph nodes	1		1				
Paravertebral lymph nodes	1						1
†Total number of deposits	124	12	51	10	31	5	15
Abdomen							
Liver	16	1	8	1	5		1
Both kidneys	11		7	1	2		1
One kidney	3	2					1
Both adrenals	9	1	4		3		1
One adrenal	5		2		1	1	1
Mesenteric lymph nodes	8	1	5		2		
Vertebrae	6		1	1	4		
Pancreas	5		2		1		2
Spleen	4	1	2		1		
Small intestine	3	1	1				1
Gall bladder	3	1	1				1
Stomach	3	1	2				
Perirenal tissue	2				1		1
Pelvic bones	2			1	1		
Peripancreatic lymph nodes	2		1		1		
Skin	2		1		1		
Peribiliary lymph nodes	1				1		
Spinal cord (lumbar)	1						1
Portal vein	1	1					
Ureter	1		1				
Peritoneum	1		1				
Lumbar veins	1		1				
†Total number of deposits	110	11	51	5	29	1	13
Femur	1			1			
No metastases‡	2				2		

*Brain was examined in ten cases.

†The difference in the sums of the columns and the number of metastatic deposits is based upon the fact that metastatic involvement of both of the paired organs (kidney, adrenal, lung, etc.) was considered as *two metastatic deposits.*

‡Brain not examined in either case.

formed, multinucleated, giant cells. In some of the tumors the giant cells formed a very conspicuous part of the histologic picture (Fig. 7). Furthermore, morphologic gradations from one form of tumor cell to another could be found in some of the tumors. These transitional forms were particularly striking in tumors exhibiting both cylindric and squamous cell varieties and suggested the possibility of a common genetic origin for all the various forms of bronchiogenic carcinomas.

METASTASIS

The tendency toward widely disseminated metastatic deposits in carcinoma of the lung has often been pointed out. The size of the primary growth bears little relation to the extent of these deposits. As will be shown later, there seems to be a relationship between the degree of differentiation and the extent of metastasis. In thirty-four (85 per cent) of the cases the tracheobronchial lymph nodes were involved; in sixteen (40 per cent) there was metastasis to the liver; in fourteen (35 per cent), there was adrenal and kidney involvement; in twelve (30 per cent), there was extension of the growth from the bronchus into the lung parenchyma; and in ten (25 per cent), both lungs were involved. In the ten cases in which the brain was examined at necropsy, six (60 per cent) showed secondary intracranial deposits. The other sites of metastasis are shown in Table II.

An analysis of Table II shows that metastatic involvement, with respect to the degree of differentiation of the parent tumor, is more frequently encountered in the adenocarcinoma than in the other types, and is least common in the oat cell variety. In the oat cell carcinoma there were six sites of metastatic deposit, or three deposits for each of the two cases, whereas in the adenocarcinoma cases there were eight metastatic deposits for each of the fourteen cases. Calculated on the basis of the number of cases in each group, the average number of metastases per case is noted:

MORPHOLOGY	NUMBER OF CASES	NUMBER OF METASTASES	AVERAGE NUMBER OF METASTASES PER CASE
Squamous cell	4	23	5.8
Oat cell	2	6	3.0
Round cell	12	62	5.2
Medullary	3	19	6.3
Adenocarcinoma	14	113	8.0
Pleomorphic	5	32	6.4

An analysis of the intrathoracic metastases shows that one of the most malignant varieties is again the adenocarcinoma, and that the oat cell and round cell carcinomas are of an approximately equal degree of malignancy; the latter two forms are apparently the least malignant. The intrathoracic metastases are as follows:

MORPHOLOGY	NUMBER OF CASES	NUMBER OF METASTASES	AVERAGE NUMBER OF METASTASES PER CASE
Squamous cell	4	12	3.0
Oat cell	2	5	2.5
Round cell	12	31	2.6
Medullary	3	10	3.3
Adenocarcinoma	14	51	3.7
Pleomorphic	5	15	3.0

At the same time it is noted that extrathoracic secondary deposits are more common in the adenocarcinoma than in the other forms of tumors. The extrathoracic metastases were as follows:

MORPHOLOGY	NUMBER OF CASES	NUMBER OF METASTASES	AVERAGE NUMBER OF METASTASES PER CASE
Squamous cell	4	11	2.8
Oat cell	2	1	0.5
Round cell	12	31	2.5
Medullary	3	9	3.0
Adenocarcinoma	14	62	4.4
Pleomorphic	5	17	3.4

While it is felt that this series of cases is too small to permit of any absolute mathematical deductions, it does support the observation that the tendency to metastasize is more common in the adenocarcinomas and least common in the oat cell variety. It also suggests the conclusion that the oat cell, round cell, and squamous cell carcinomas tend to spread by contiguity and via the lymphatics, whereas the medullary and adenocarcinomas spread by way of the blood stream. It must, of course, be borne in mind that the very location of these growths (with respect to the vascular system) may lead to a rapid and distant spread of the disease once it has broken into a blood vessel, regardless of the cellular characteristics of the mother growth. The foregoing is in accord with the views expressed by Samson who believes that there is a relationship between the histopathology of the primary tumor and the sites of metastatic deposition. In the pleomorphic cell carcinoma cases the mode of spread was apparently similar to that of the most malignant cell type comprising the tumor.

DISCUSSION

The adherents to the unicellular theory of tumor origin believe that the different forms of growth that may arise from a particular tissue or organ of the body must, of necessity, have a common genetic origin. Histologically it is noted that the bronchial mucosa is lined by three different types of cells, namely, ciliated, goblet, and basal cells. Of these three types of cells, the ciliated and goblet cells are the most differentiated, while the basal cells are the least differentiated and, therefore, the most primitive in nature. Tuttle and Womack

have shown that the cells lining the bronchial mucous glands also have their origin in the bronchial mucosal epithelium. It seems very unlikely that fully differentiated cells, such as the ciliated and goblet cells, would revert through cell metaplasia to other forms of epithelial tumors such as the squamous cell variety. It seems also unlikely that these differentiated cells, when undergoing malignant changes, should acquire various forms of cell characteristics which are relatively foreign to them. An explanation for such changes must, therefore, be sought along other more plausible lines. Fried, among many others, is also of the opinion that all bronchiogenic carcinomas have their origin in the basal cell layer of the bronchial wall.

It was Krompecher who first advanced the theory that the basal cells lining the depth of the mucosa of certain organs, viz., stomach, appendix, colon, uterus, etc., are multipotent and capable of giving rise to glandular and cylindric epithelium. Under certain pathologic conditions these so-called basal cells are able to form either flat epithelium, cylindric epithelium, or both. Cell metaplasia, according to Krompecher, is therefore not a result of postembryonic change in the histologic appearance of the cell, but is due to the differentiation of the basal cells into either squamous or cylindric epithelium as the case may be. Roessiger supports this theory and finds it to hold true in explaining certain metaplastic changes that sometimes occur in the gall bladder; Rabinovitch finds it to hold true in the case of the gall bladder and kidney, and Teutschlander holds that the same may occur in the respiratory tract.

The histopathologic findings noted in our series show several distinct tumor cell varieties; it also seems evident that these different forms of tumor cells have originated from the deeper basal cell layer lining the mucosa of the bronchial tree. This conclusion is based primarily on the fact that the basal layer of epithelium is the least differentiated and possesses the various potentialities for growth and cell differentiation. Such a conclusion would also confirm the belief that the various epithelial growths have, in all probability, a common genetic origin and that they are all capable of responding to the same growth stimulant. If one is to conceive the process of tumor cell differentiation in this light, then one can state definitely that the different configurations in these various forms of growth are not separate and distinct tumor cell entities but different morphologic manifestations of growth of one and the same tumor type. In other words, one is probably dealing in these instances with tumors arising from a common type of cell which, under the influence of the irritant, has acquired more than one histologic cell characteristic. According to this assumption, the development of histologically different kinds of neoplasm in the same organ is rendered possible through the differentiation of the primitive

basal cell into its varied forms of growth. In a number of specimens in our series, especially in the pleomorphic forms of bronchiogenic carcinoma, such transitional changes from one form of carcinoma to another were quite apparent. Thus, on examining numerous fields of the same section, one could find many intermediary stages of tumor cell development. In certain areas the neoplasm exhibited features peculiar to the squamous cell carcinoma, and in others, it resembled cylindric cell or glandular carcinoma. In still other instances the tumor was found in a transitional phase of cell development. In the latter case the cells appeared as nests of flat epithelium closely resembling the squamous cell carcinoma, but differing from the latter in that there was a lack of prickling or pearl formation. Some investigators have referred to this transitional form of growth as "pseudosquamous carcinoma" while others have included it under the general heading of squamous cell carcinoma. We, however, have not chosen to regard it as either pseudosquamous or squamous cell carcinoma and have classified it as a pleomorphic variety of bronchiogenic carcinoma. This probably explains the relatively large number of squamous cell carcinomas reported by other investigators as compared with our series. In reality these so-called pseudosquamous carcinomas of the lung merely represent an arrest in the further differentiation of the tumor cell, and as such, cannot be regarded as either squamous or pseudosquamous carcinomas. The same holds true for the other forms of pulmonary cancers which show a varied combination of cell types, such as the round cell, oat cell, medullary form, etc. Womack and Graham have described a potentially malignant group of mixed tumors of the lung which histologically resemble fetal pulmonary tissue. In many instances neoplastic cartilaginous and osseous tissue was found in the stroma of the growth. No such findings were noted in any of our cases of bronchiogenic carcinoma. We are of the opinion, therefore, that the various forms of cancer of the bronchus described in the literature are no more than different manifestations in the developmental phase of one common type of tumor cell. Any attempts made to separate these various forms of growth according to their morphologic cell characteristics has caused considerable confusion in the past, especially when one attempted to identify them as separate tumor cell entities. We feel that the classification of these tumors, as presented in this paper, meets with all the requirements enumerated above and includes all the forms of primary carcinoma known to occur in the bronchial tree.

CONCLUSIONS

A pathologic study of forty cases of bronchiogenic carcinoma with necropsy observations is presented.

A classification of these tumors is offered to include all the forms of primary malignant neoplasia which may be encountered in the bronchial tree.

The unicellular theory seems to hold true in explaining the various forms of malignant growths that may arise in the bronchial tree.

REFERENCES

1. Fried, B. M.: Primary Carcinoma of the Lung, Medicine **10**: 373, 1931.
2. Ewing, J.: Neoplastic Diseases, Philadelphia, 1931, W. B. Saunders Co.
3. Hochberg, L. A., and Lederer, M.: The Early Manifestations of Primary Carcinoma of the Lung, Arch. Int. Med. **63**: 80, 1939.
4. Hochberg, L. A.: Roentgenographic Images in Primary Carcinoma of the Lung, Radiology **32**: 713, 1939.
5. Hochberg, L. A., and Welt, B.: Primary Carcinoma of the Lung From the Bronchoscopist's Point of View, M. Rec. **150**: 265, 1939.
6. Krompecher, E.: Basalzellen, Metaplasia und Regeneration, Beitr. z. path. Anat. u. z. allg. Path. **72**: 163, 1924.
7. Lumsden, C. E.: Pulmonary Carcinoma; a Pathological Study of a Series of Cases With Special Reference to the Route of Spread and to the Factors That Determine the Mode of Spread, Glasgow M. J. **131**: 57, 1939.
8. Rabinovitch, J.: Squamous Cell Carcinoma of the Kidney, Arch. Surg. **24**: 581, 1932.
9. Rabinovitch, J., and Kieffer, R. S.: Squamous Cell Carcinoma of the Gall Bladder, Surg., Gynec. & Obst. **52**: 831, 1932.
10. Roessiger, W.: Ueber Mischkrebse der Gallenblase, Beitr. z. path. Anat. u. z. allg. Path. **85**: 181, 1930.
11. Samson, P. C.: Relation of Cell Type to Metastasis in Bronchogenic Carcinoma, Am. J. Cancer **23**: 745, 1935.
12. Simons, E. J.: Primary Carcinoma of the Lung, Chicago, 1937, The Year Book Publishers.
13. Teutschlander: Ueber Metaplasie und Krebsbildung, Verhandl. d. deutsch. Path. Gesellsch. 184, 1923.
14. Tuttle, W. McC., and Womack, N. A.: Bronchogenic Carcinoma; a Classification in Relation to Treatment and Prognosis, J. THORACIC SURG. **4**: 125, 1934.
15. Womack, N. A., and Graham, E. A.: Mixed Tumors of the Lung, Arch. Path. **26**: 165, 1938.

DIFFERENTIAL INTRABRONCHIAL PRESSURES AND MEDIASTINAL EMPHYSEMA

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ALTHOUGH Elsberg,[1] in 1910, claims to have performed the first intrathoracic operation using an anesthetic given by intratracheal insufflation, it is known that, as early as 1896, Tuffier[1] of Paris passed a metal tube through the larynx and anesthetized a patient in this manner, following which a partial lung resection was performed. Two years later, Parham[1] of New Orleans did a resection of the chest wall for a malignant growth. The anesthetic for this case was administered through a tube which dipped down into the trachea.

During the year following Elsberg's first intratracheal insufflation for thoracic surgery, he succeeded in giving more than 100 subsequent intratracheal anesthetics and found them very satisfactory for various types of operative procedures.[2] No untoward results were obtained, and he believed that vomiting, coughing, and expectoration were very rare following intratracheal anesthesia. He found it to be a distinct advantage where the patient must be placed on the abdomen for surgical measures.

Bradshaw[3] states quite tersely that knowledge of pulmonary physiology and pathology has been accumulating since the time of Vesalius, but that knowledge and experience in surgery of thoracic diseases are of recent origin. Sauerbruch,[4] Meyer, Lilienthal, Graham, and others have played a most important part in developing thoracic surgery.

That a patient could survive an open chest wound or operation was known many years ago; but, until recently, intratracheal positive pressure anesthesia was considered necessary for all intrathoracic operations. This has been disproved during the past six years, since many of these operations have been performed under local, spinal, or simple face mask anesthesia.

In giving intratracheal insufflation anesthetics, there are many factors which enter into a smooth operative and postoperative course. Mediastinal emphysema and pneumothorax are complications against which it is necessary to guard. Though the causative factors may be varied, the chief among them is the increased intrabronchial pressure obtained in giving the anesthetic.

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Fundamentally there is but one method in the administration of an intratracheal anesthetic. It consists of a tube being placed in the trachea and connected to a source of anesthesia. The means by which this is accomplished are many and varied, and the types of machines and appliances employed in the administration of this anesthetic are numerous. The ends achieved, however, are usually quite similar.

In the past decade intratracheal anesthesia has made tremendous advances, but it is quite apparent that research in this particular field has not reached the level necessary for uniformly satisfactory results in this type of anesthesia. Too many unexplained fatalities have occurred, necessitating further investigation.

Stephens[5] recently reported three cases dealing with this problem. He points out that rupture of the mediastinal pleura causes embarrassment of cardiac action and a change in respiration. With bilateral open pneumothorax both lungs expand poorly, heart action becomes feeble, and unless a rather forceful introduction of air into the lungs is accomplished, the patient will die of cessation of the heartbeat.

In a report on three patients upon whom thoracic surgery had been performed, two died showing a contralateral pneumothorax. At autopsy no tear in the mediastinum could be demonstrated, although it was believed that a rent had been made in it. The first of the two patients who died also developed a marked emphysema of the face, neck, and mediastinum. In both of these cases ordinary mask anesthesia technique was used. In a third case, spontaneous pneumothorax was suspected, and upon aspiration of the opposite pleural cavity, this condition was found to exist. Air was withdrawn and positive pressure administered, and consequently this patient's life was saved. In this case both intratracheal and mask procedures were utilized.

In the above three cases it is possible that a rupture of the alveoli caused the pneumothorax, but it is believed by the authors that leakage of air occurred through the mediastinum. However, from the studies made by Escudero and Adams,[6] it has been concluded that simple overdistention of the remaining lung tissue, complementary to massive atelectasis, is sufficient for the development of spontaneous pneumothorax. The air in the pleural cavity would occur by quite a different route and will be discussed later.

We have in our records the case of a patient upon whom a preganglionic, upper thoracic sympathectomy was performed, during which a right pneumothorax accidentally occurred. When the pleura was being closed, the lungs were distended with a pressure of 12 mm. mercury until the opening was made airtight. Fourteen hours later marked subcutaneous emphysema occurred over the neck and face, extending below the scapula and over the right temple. Roentgenograms taken at this time disclosed a pneumothorax on the left unoperated side with complete collapse of the left lung but no mediasti-

nal shift. Aspiration of air from the left pleural cavity gave relief of dyspnea, and the emphysema gradually disappeared. Recovery thereafter was uneventful.

Bradshaw[3] has reported a case of an 18-month-old female child who was given an intratracheal anesthetic for the removal of an intrathoracic neurofibroma. Shortly after the anesthetic was begun, subcutaneous emphysema of the neck was noticed. The anesthetic was stopped immediately, and x-rays of the chest showed a small right pneumothorax. The operation was performed some days later with simple mask anesthesia. Bradshaw stresses consideration of the size of the lumen of the trachea in small children and infants, pointing out that it is very easy to overdistend the lungs due to the very small outlet. The pneumothorax and emphysema in both of the foregoing cases was undoubtedly due to overdistention of the lungs, thus leading to mediastinal emphysema which gave rise to the above complications.

Eisenbrey,[7] in 1912, made observations on the use of intratracheal anesthesia in experimental work and concluded that the intrabronchial pressure was affected to but a slight degree by the pressure which is forced into the lungs. He found that a pressure of 20 mm. of mercury going into the trachea gave only 1.4 mm. of mercury of intratracheal pressure. When the ingoing pressure was raised to 50 mm. of mercury, the intratracheal pressure was only 2.2 mm. of mercury. However, any constriction of the outflow raised the pressure to a marked degree. He states further that any pressure sufficient to cause a cessation of respiration causes a fall in blood pressure of from 20 to 40 mm. of mercury. When the pressure is relieved, respiratory movements are immediately resumed.

In our experimental work we have substantiated the above findings. Our interest in this problem was aroused by the death of several animals following operations on the chest under intratracheal anesthesia. Autopsy revealed that death in these cases was due to collapse of the lungs and to pneumothorax.

In our studies we have measured ingoing pressures, intratracheal pressures, and changes in blood pressures and in respiratory movements. One of our chief considerations was to determine the amount of intrabronchial pressure necessary to cause mediastinal emphysema.

It is well known that there are methods of causing mediastinal emphysema other than that of increased intrabronchial pressures. In a recent report by Banyai and Jurgens,[8] it was stated that, out of forty-two cases of subphrenic pneumoperitoneum, seven developed mediastinal emphysema. In some it occurred within a few minutes, and in others, not for several hours. It was characterized by pain about the larynx, choking, coughing, and shoulder pain. Complete relief was obtained in from one hour to four days. In all but one of the cases there was a palpable emphysema of the neck.

It is believed by the authors that the air escapes from the peritoneal cavity and reaches the mediastinum in one of two ways. It may spread along the large vessels or esophagus and then into the mediastinum, or it may pass through the peripheral portion of the diaphragm which might be traumatized by the needle. A roentgenogram is the best means of confirming the diagnosis. In all of the seven cases, a peritoneal pressure of +12 to +18 cm. of water obtained.

One patient who was seen in the University of Chicago Clinics developed a severe emphysema of the neck and face following a prolonged period of asthmatic coughing. When the attack was controlled, the emphysema subsided. In this case there was no evidence of pneumothorax.

In our first few experiments we wished especially to note the effect of increased intrabronchial pressure on blood pressure and respiratory rate and volume. An intratracheal catheter was employed, delivering a continuous stream of air and ether to the lower end of the trachea. The outlet for the air in the lungs was through a tube (connected with a tightly fitted face mask) which led beneath the surface of water. Thus, the intrabronchial pressure was regulated by the amount of resistance to escaping air from the lungs.

Maximum intrabronchial pressures in the various experiments on dogs varied from 16 to 110 mm. of mercury. The accompanying fall in blood pressure varied from 8 points with a 6 mm. rise in intrabronchial pressure to 98 points with a 100 mm. rise in intrabronchial pressure. Pressures of 24 mm. of mercury or over were routinely accompanied by development of emphysema, usually only along the great vessels at the hilum of the lungs. On two occasions, however, emphysema spread up along the trachea into the neck. In one animal intrabronchial pressure of only 18 mm. of mercury produced emphysema.

Table I illustrates the changes in blood pressure and respiration caused by increased intrabronchial pressures. It also shows the pressures necessary to initiate emphysema and the degree to which it developed.

In a series of five cats it was found that considerably less pressure was necessary for the production of emphysema. Without exception, pressures of 16 to 20 mm. of mercury resulted in emphysema. This may be due in part to the mediastinal structures of the cat which differ from those of the dog in that the superior mediastinum (in the cat) has a double layer of pleura and considerable substance. In the dog, the air bubbles travel along the vessels which course through the mediastinum and eventually reach the neck. In the cat, the potential space between the double layer of pleura and the fatty tissue within the superior mediastinum lends itself very well to permeation of air due to increased pressure, and at autopsy it was observed that the superior mediastinum was filled with air. Fig. 1 is a photograph taken

TABLE I

INTRABRONCHIAL POSITIVE PRESSURE STUDIES

DOG	DATE	INTRABRONCHIAL PRESSURES			BLOOD PRESSURES		RESPIRATORY EFFECT		MEDIASTINAL EMPHYSEMA DEGREE AND REMARKS
		AVERAGE MM. HG	MAXIMUM MM. HG	DURATION IN MINUTES OF INCREASED PRESSURE	FALL MM./HG	INCREASE IN INTRA-BRONCHIAL PRESSURE	IMMEDIATE	LATE	
1	8/25/38	6-14	18	43	16	14	Apnea	Normal	Slight emphysema developed after chest was opened
2	9/ 8/38	8-14	16	18	20	16	Apnea, 35 sec.	Decreased rate	None
3	9/20/38	20-24	26	60	32	26	Apnea	Decrease in volume	Extensive emphysema at hilum and into neck
4	9/27/38	30-44	44	25	Not recorded	Not recorded	Chest opened	Chest opened	Emphysema developed after chest was opened and pressure put up to 30 mm. Hg
5	9/29/38	12-28	28	75	8	6	Apnea	Normal	Small amount in hilum
6	10/ 6/38	25	25	25	14	8	Apnea	Normal	Moderate along vessels to right lower lobe
7	10/11/38	24	24	30	Slight	Gradual	Apnea	Normal	Marked emphysema but no evidence of rupture
8	10/13/38	20	20	30	22	18	Apnea	Decreased rate	None
9	10/18/38	20	20	30	28	20	Apnea	Decreased rate	None
10	3/21/39	80-110	110	50	98	110	No resps.	Few resps.	Extensive into neck and at lung periphery; no rupture
11	3/22/39	80-100	100	35	84	92	Apnea	Diminished	Extensive at hilum; dead in 18 hours; hemothorax

Ether anesthesia used in all experiments. All dogs given morphine sulfate previous to anesthetic.

after autopsy of a cat which had been subjected to a pressure of 20 mm. of mercury for a period of twenty-five minutes. Air had spread up along the trachea into the neck and along the vessels into the axillae.

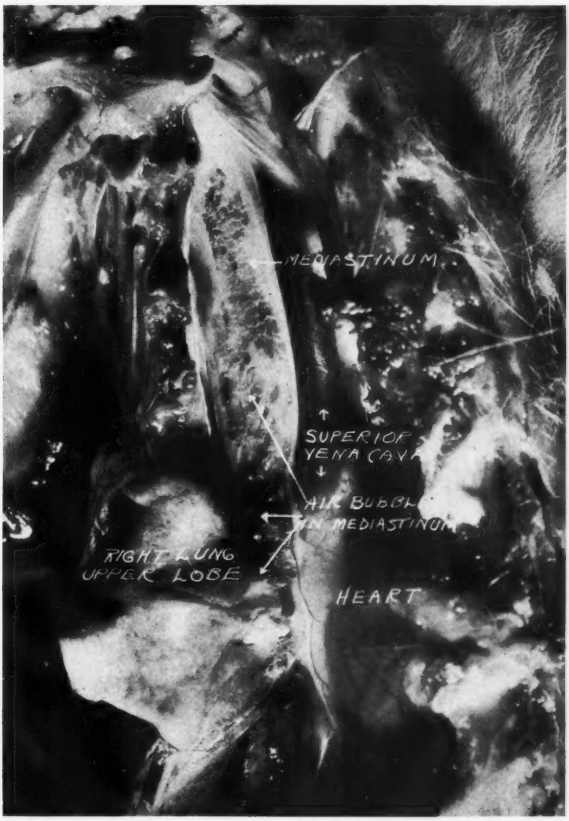

Fig. 1.—Photograph taken immediately after opening the thoracic cage of a cat subjected to 20 mm. of mercury intrabronchial pressure for twenty-five minutes. It will be noted that the superior mediastinum is filled with small air pockets and that the emphysema has spread along all the vessels at the roots of the lungs, leading eventually to the mediastinum.

Fig. 2 is a kymographic tracing showing the changes in blood pressure in a dog due to an intrabronchial pressure of 14 mm. of mercury. The two-minute period of apnea and the slowing in respiratory rate are clearly seen.

Fig. 3 demonstrates the marked fall in blood pressure bordering on right cardiac failure due to a seven-minute period of markedly increased intrabronchial pressure which averaged approximately 55 mm. of mercury.

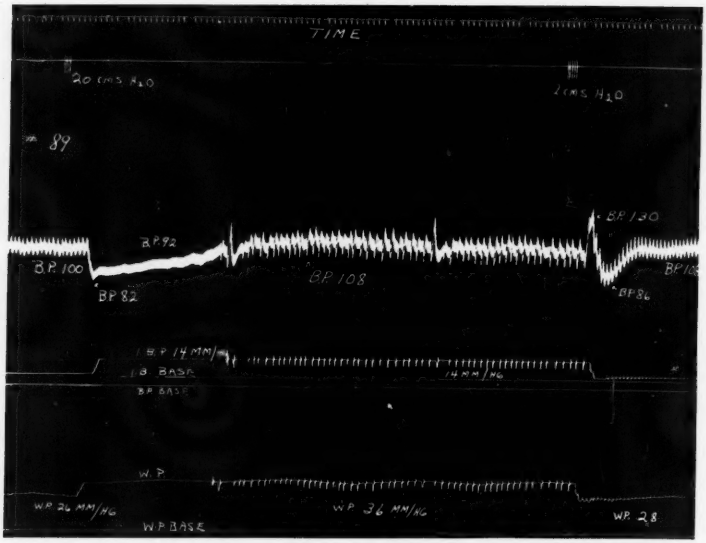

Fig. 2.—Kymographic tracing showing the decrease in blood pressure and the period of apnea due to a rise to 14 mm. of mercury intrabronchial pressure. When the pressure is relieved, there is seen a transitory rise of the blood pressure above normal, and finally the normal level is reached. Respirations which were decreased during the increased intratracheal pressure are seen to become more rapid when the pressure was removed. (B.P. = blood pressure; I.B.P. = intrabronchial pressure; W.P. = wall pressure.)

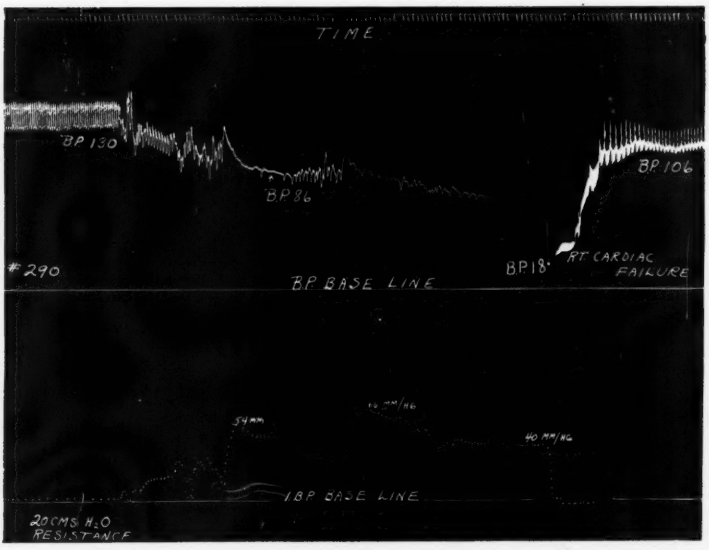

Fig. 3.—Kymographic tracing showing the continued fall in blood pressure resulting in right cardiac failure due to markedly increased intrabronchial pressure. (B.P. = blood pressure; I.B.P. = intrabronchial pressure.)

Macklin[9] has performed rather extensive research on this subject. His work has been confined chiefly to overinflation of isolated lobes of the lungs by using small catheters which fit well down into the bronchus. Pressures which he used varied from 10 to 220 mm. of mercury. This is not the true intrabronchial pressure as far as is known, but rather the ingoing pressures. However, with the outflow limited by a tightly fitting catheter, the intrabronchial pressure may quite closely approximate these figures.

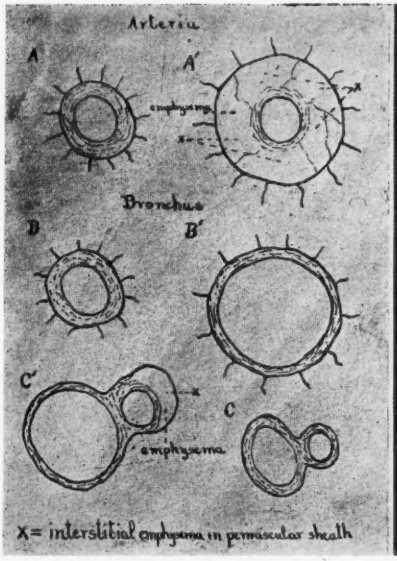

Fig. 4.

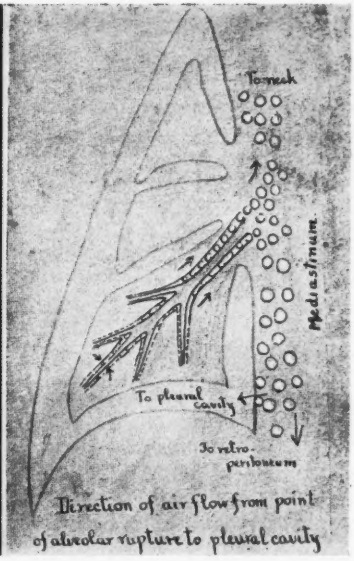
To neck
Mediastinum
To pleural cavity
To retro-peritoneum
Direction of air flow from point of alveolar rupture to pleural cavity

Fig. 5.

Fig. 4.—Diagram to show swelling of sheath of pulmonary arterial branches in the insufflated lobe, in contrast to the thinning of the wall of the air tubes under the same conditions. *A*, *B*, and *C* represent the approximate normal, and *A'*, *B'*, and *C'* show the insufflated condition. Note the thickening of the arterial sheath in *A'* as contrasted with the normal sheath in *A*, and also the invasion of air *X*. The air tube sheath in *B'* is thinner than that of the normal, and its substance supports the alveolar bases, shown between the radiating partitions; in the case of the artery, the alveolar bases are deprived of their normal support on account of the change in the underlying tissue of the sheath. *C* represents both artery and bronchiole, and their condition in insufflation is shown in C'. (After C. C. Macklin.[9])

Fig. 5—Diagram showing the pathway of air from the air spaces of the lung to the mediastinum and beyond, via the perivascular sheaths of the lung. Only the artery is represented. Bubbles of air in the sheaths and connective tissue of the mediastinum are shown as high-lighted spherules. One of the many points of invasion of the perivascular sheaths is indicated by the arrows in the lung interior; and one of the sites of break-through into the pleural cavity is also indicated by an arrow. (After C. C. Macklin.[9])

It is believed by Macklin that, in lobes which are overinflated, the air in finely subdivided form breaks through from the alveoli abutting upon the perivascular sheaths into the perivascular tissue which has become swollen and succulent and undoubtedly weakened through the inhibition of fluid. This "break-through" occurs during insufflation

when that portion of lung is overdistended, and a pressure gradient presumably exists from the distended alveoli to the underlying connective tissue.

Figs. 4 and 5 are diagrams taken from Macklin's work, which show the course taken by the air or gases in the production of mediastinal emphysema and other complications resulting from overdistention of the lung by increased intratracheal pressures. Undoubtedly, this is the most reasonable and logical explanation offered relative to the above complications.

Macklin[10] has recently been able to demonstrate tears through which air had passed from the alveoli into the perivascular sheaths. Globules of air in the space which they previously occupied frequently have been found in the sheaths, into which they have undoubtedly broken through from the alveolar bases. Collectively the boundaries of the sheaths of the insufflated region would form a considerable area through which air could leak into the interstitial tissue, and which would be large enough to account for the large volume of air which accumulated in the mediastinum and beyond.

Macklin, however, has not pointed out that, once the chest has been opened and increased intrabronchial pressure applied, emphysema occurs more easily, as there is then greater stretching of the perivascular tissues with less pressure because of removal of the restraining chest wall. In addition, we have found that, once emphyema has begun, the pressure necessary for its continued development is somewhat less than that required to initiate it.

In view of the prominent role which intratracheal anesthesia has received during the past decade, it seems that too often it is being employed in association with increased intratracheal pressure, where a less hazardous technique would suffice. Many clinics regard positive pressure as indispensable, but, if we are to decrease the complications which too often terminate fatally, it is quite imperative that the cases be well selected and that the administration of this type of anesthesia be handled by those who are competent, capable, and with a knowledge of the hazards involved. At all times we should be on the alert for complications, and, if they occur or are suspected, the necessary therapeutic measures should be instituted immediately.

CONCLUSIONS

Intratracheal or positive pressure anesthesia may result in serious or fatal complications.

Increased intrabronchial pressure either by intratracheal or mask anesthesia technique may produce deleterious effects by overdistention and laceration of the intrapulmonary tissue with subsequent development of mediastinal emphysema and pneumothorax.

Intrabronchial pressure depends chiefly upon the patency of the outlet, rather than upon the pressure at which the gases are forced into the lungs.

Intrabronchial pressures of 18 mm. of mercury or below seldom, if ever, cause mediastinal emphysema in dogs.

Extremely high intrabronchial pressures are necessary to cause emphysema about the neck and face or to cause right cardiac failure in dogs.

Pneumothorax may easily occur following mediastinal emphysema.

Once emphysema has developed, it may be increased at a pressure lower than that necessary to initiate it.

REFERENCES

1. Elsberg, Charles A.: Clinical Experiences With Intratracheal Insufflation (Meltzer), With Remarks Upon the Method for Thoracic Surgery, Ann. Surg. **52**: 23, 1910.
2. Elsberg, Charles A.: Further Experiences With Anesthesia by the Intratracheal Insufflation of Air and Ether, Ann. Surg. **53**: 749, 1911.
3. Bradshaw, H. H.: Anesthesia for Intrathoracic Operations, J. THORACIC SURG. **8**: 293, 1939.
4. Killian, Hans: Scope and Utility of Differential Pressure in Thoracic Surgery, Current Researches in Anesth. & Analg. **17**: 146, 1938.
5. Stephens, H. Brodie: A Consideration of Contralateral Pneumothorax as a Complication of Intrathoracic Operations, J. THORACIC SURG. **5**: 471, 1936.
6. Escudero, L., and Adams, W. E.: Spontaneous Pneumothorax Associated With Massive Atelectasis, Arch. Int. Med. **63**: 29, 1939.
7. Eisenbrey, Arthur B.: Observations on the Use of Intratracheal Anesthesia in Experimental Work, Surg., Gynec., & Obst. **15**: 715, 1912.
8. Banyai, A. L., and Jurgens, G. H.: Mediastinal Emphysema as a Complication of Artificial Pneumoperitoneum, J. THORACIC SURG. **8**: 329, 1939.
9. Macklin, C. C.: Pneumothorax With Massive Collapse From Experimental Local Over-Inflation of the Lung Substance, Canad. M. A. J. **36**: 414, 1937.
10. Macklin, C. C.: Transport of Air Along Sheaths of Pulmonic Blood Vessels From Alveoli to Mediastinum, Arch. Int. Med. **64**: 913, 1939.

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The 1940 Meeting of the American Association for Thoracic Surgery
Notice to Members

The headquarters for the meeting in Cleveland will be the Wade Park Manor, and members will shortly receive information from the management about reservations.

The combined meeting with the National Tuberculosis Association on June 6 will be held in the Hotel Statler. The meetings on June 7 and 8 will be held in the Academy of Medicine, which is near the Wade Park Manor.

The program will be closed on March 1, and no papers will be accepted after that date. This date also applies to the papers submitted for the Rose Lampert Graff Foundation prize. Abstracts and papers should be sent to the secretary as soon as possible.

RICHARD H. MEADE, JR.,
Secretary.